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ON

EPILEPSY:

ANATOMO-PATHOLOGICAL AND CLINICAL NOTES.

(WITH ORIGINAL PLATES AND ENGRAVINGS.)

BY

M. GONZALEZ ECHEVERRIA, M.D. (Univ. Paris),

PROFESSOR OF MENTAL AND NERVOUS DISEASES AT THE UNIVERSITY MEDICAL COLLEGE OF NEW YORK; PHYSICIAN IN CHIEF TO THE NEW YORK HOSPITAL FOR EPILEPTICS AND PARALYTICS; VISITING PHYSICIAN TO CHARITY HOSPITAL, BLACKWELL'S ISLAND, N. Y., ETC., ETC.

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TO

DAVID LYMAN, ESQ.,

AS

A SINCERE TOKEN

OF

THE AUTHOR'S AFFECTION.

PREFACE.

The object of this book is manifest by its title. It does not claim to offer any new discovery, although it is possible that it may shed some light on subjects not well established. Still less is it designed to be an English treatise on Epilepsy: classical for this purpose are the works of Sieveking, Radcliffe, Reynolds, and the lectures of Trousseau and of Brown-Séquard, full of science, and in a form suited to the wants of teacher and learner. I have attempted to meet a deficiency in my own knowledge by comparing the cases of epilepsy I have carefully noted during the past ten years; how far my notes meet the wants of others remains to be seen. It is more than a year since most of these researches went to press. Unavoidable circumstances, and the printing of the chromo-lithographs illustrating the text, have delayed their appearance beyond what I contemplated. Further opportunities of inquiry have occurred in the mean time, giving greater strength to my conclusions. Five additional autopsies of epileptics have regularly displayed the alterations I have pointed out in the sympathetic. The lesion is constituted mainly by a proliferation of connective elements, and their subsequent substitution to the nerve-cells and fibres, finally undergoing retrograde degeneration. The change exhibits itself, in various stages, throughout the nervous plexus, around the vertebral and cerebral arteries, and in the cervical sympathetic. It operates essentially in the alterations of the capillaries in the brain and medulla, and I am not aware of any mention made of such a lesion of the sympathetic as proper to epilepsy.

These being personal notes, I have, out of conciseness, not referred in every instance to doctrines or facts established by other authors; and I wish that this acknowledgment will suffice for all cases in which no mention might be made of their researches. The plates are faithfully copied by myself from specimens I have carefully prepared, and which have been also examined by my Assistant Physicians, my pupils, and many of my colleagues.

No better opportunity will offer itself for stating my gratitude to my esteemed friend, Professor Charles Robin, of Paris, with whom I have spent many hours of the pleasantest recollection, and to whom belongs any credit that my investigations might deserve. To the satisfaction of counting myself among his pupils, to many warm proofs of predilection and friendship, I still add the practical teaching which has served as the most secure guide in my inquiries on epilepsy and other nervous diseases.

In conclusion, I must own particularly the valuable aid I have derived respectively from my Assistants, Drs. F. A. Castle, L. B. Edwards, F. M. McClung, D. McEwan, J. H. Morgan, A. E. Macdonald, and W. H. Bennett. Each of them shares importantly in my work, and I am most happy to express this unqualified acknowledgment of their efficiency and talents. To Messrs. Isaac Bell, James Owen, James B. Nicholson, O. W. Brennan, and A. Frear, of the Board of Commissioners of Public Charities and Correction, I feel also much obliged for their worthy and unabating efforts to keep the New York Hospital for Epileptics and Paralytics on a model standard, no less than for their kindness and consideration.

50 West Thirty-third Street,
New York, *October*, 1870.

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EPILEPSY.

CHAPTER I.

MODERN DOCTRINES OF EPILEPSY.

Epilepsy is a name, synonymous with convulsion, indiscriminately employed by ancient and modern medical writers to designate different spasmodic affections. No other malady exhibits a wider range in its etiology, and this plainly accounts for the above confusing application. There is scarcely a disease deranging the human frame, in which epileptiform convulsions might not happen as an incident or essential phenomenon, and it may be safely set down as a truth of great importance, that the numerous conditions capable of inducing epilepsy give to each of its species a characteristic impression that will ever prevent conforming their individual to any typical case, or finding any specific cure for every instance of the disease. To establish the peculiar morbid conditions influencing its development, to discriminate the general from the local circumstances, in order to arrive at a rational and successful treatment, is the fundamental question in the study of epilepsy.

Characteristic as many of the groups are, we cannot consider them alone, and the study must merely

refer to the so-called idiopathic epilepsy, and leave out of view entirely the epileptiform convulsions incident to disease generally. Let it be, however, said in making this distinction, that under the head of idiopathic are classified those cases in which the spasmodic paroxysms, at first induced by some systemic or local lesion, have afterwards continued as *a habit*, notwithstanding the disappearance of their original cause, until they became an individuality the product of action and reaction. In addition, the morbid phenomena of idiopathic epilepsy should by no means be regarded irrespective of organic trouble, namely, that the disease is idiopathic because no structural changes of the nervous system were found associated with it. Epilepsy, like paralysis, is not a morbid entity, existing by itself, but a manifestation of manifold material derangements disturbing the nervous system and giving rise to a definite inseparate condition — immediate cause of the convulsive paroxysm — that remains the same whatever be the occasional origin of epilepsy.

Since the earliest ages of medicine, epilepsy has been considered as an essential affection of the brain, and its source successively ascribed to almost every conceivable cause, from the demoniacal to the no less mysterious cerebral irritation of our days. It was reserved for the great activity of experimental physiologists to throw the light that fertilizing the barren field has brought forth more fruitful results on such a sterile one as epilepsy. But, with all this advance-

ment, there remain still many obscure questions connected with the subject, which have to be decided by anatomo-pathological rather than by physiological or clinical inquiries. Frequent, important, and dreadful as are the instances of epilepsy, there has been yet no settled opinion concerning the pathological anatomy of the disease. With this idea in view, I have kept a careful record of every case of epilepsy in the hospitals, or in private practice, which has come under my observation during the past eight years, investigating as accurately as possible the circumstances related to it, and availing myself of every opportunity to study the dead bodies of epileptics in order to compare the results of these cadaveric investigations and those of vivisections; while the facts so registered have either not supported the erroneous anticipations of some authors, or have overturned the foundations of previously established hypotheses, they yet serve to confirm the soundness of newer doctrines. Their evidence may be perhaps judged insufficient— I will not question it — but it suffices to substantiate the opinions here drawn in relation to the important subjects connected with this matter. Nevertheless, far from me to attempt to pass an absolute verdict on any of such doctrines, or to urge an improved one as voucher for a novel treatment. I only relate what I have observed, and it supports the convictions here averred. On maintaining them, frequent allusion has to be made to the modern theories of epilepsy. An outline of their essential points will, therefore, contri-

bute to a better understanding of my assertions, as it would be too long and inexpedient to analyze them in detail. Such a summary, however, is not calculated to present original interpretations of the facts therein contained, but to trace through what successive steps experimental physiology and pathological anatomy have to a considerable extent confirmed the correctness of views, most of them, intuitively anticipated by an imperfect science.

The frequency with which those who have investigated the seat and cause of epilepsy have confounded them, accounts for the unsettled opinions on the subject. Pathological investigations and vivisections have decided that there are portions of the nervous centres principally involved in epilepsy, and that in every case they display in a higher or lesser degree a material modification, which may or may not be associated with other organic alterations. This has so far been established, and in such organic modification — always identical — lies the reason why the disease does not vary with the many circumstances that may engender it.

The reflex power of the spinal cord, or rather of the medulla oblongata, and derangement of the cerebral circulation, are the two elements which, separate or together, constitute the base of our leading doctrines of epilepsy.

Marshall Hall, whose name will ever remain associated with our advancement in the knowledge of nervous diseases, and especially of epilepsy, was the

first who tried to explain the unintelligible origin of the paroxysmal diseases of the nervous centres by the action of the spinal or diastaltic nervous system.* He established: "a class of diseases of the nervous centres, inorganic in their origin, and paroxysmal or recurrent in their form, including not only apoplexy and paralysis, but these and epilepsy, and mania, as one and the same disease differing in degree. But whilst apoplexy affects the cerebrum, and paralysis a hemisphere, epilepsy affects the medulla oblongata, and mania again the cerebrum. In speaking of these diseases we may now speak not of apoplexy, paralysis, epilepsy, mania, but of nervous seizures, assuming an apoplectic, paralytic, epileptoid, or maniacal form; and thus our diagnosis will be implied in one designation. Indeed, these affections pass into each other. The causes of paroxysmal seizures are: direct or centric—the emotions, and diastaltic or reflex—or excentric irritations. All these causes act on the oblong medulla, which is the centre of the excito-motory power. The excitation of this centre exhausts itself upon being called to action, but gradually regenerates itself again, thus accounting for the cessation and occasional recurrence of the seizures, which if too great, completely exhausts the excito-motory power and strength of the patient, who remains nevertheless susceptible to renewed attacks." "The emotions act through the

* Synopsis of Cerebral and Spinal Seizures of Inorganic Origin and of Paroxysmal Form.—Second edition of an outline of the Croonian Lectures delivered at the Royal College of Physicians in 1851.

nerves of the muscles of the neck *directly*. The irritations act on the same muscles *diastaltically*. Why these causes should select the muscles of the throat principally for the display of their influence, is a mystery, but it is not less the fact that they do so; the medulla oblongata, however, is the centre of the diastaltic action and of the larynx. The primary seat of these actions must be viewed as two-fold: 1st, the neck generally; 2d, the larynx specially; according as the neck only, or the larynx is affected—1st, *trachelismus;* or, 2d, *laryngismus* is produced. As the condition of the neck and larynx exists in two forms or degrees of intensity, so do their effects, and apoplexy and epilepsy are seen in the forms: 1st, of *apoplexia mitior*, and *gravior;* and 2d, *epilepsia mitior* and *gravior;* the milder form being the effect of trachelismus, the severer of laryngismus. But, if the trachelismus pass into laryngismus, the apoplexia mitior assumes the form of apoplexia gravior, and the epilepsia mitior or the "*petit mal*" of the French authors, that of epilepsia gravior or the "*grand mal*" or "*haut mal*" of the same writers. If the affection does not advance beyond trachelismus, the appearance and symptoms may be subdued by venesection. If laryngismus has taken place, the symptoms and appearances are subdued by tracheotomy." Let it be added, that—"epileptic attacks, like every direct or reflex action of the spinal system, are *always* excited."*

* Aperçu du Système Spinal, etc. Paris, 1855, p. 138.

Marshall Hall, besides, was the first to notice particularly the correspondence of loss of blood and epileptic attacks in general, and to adduce facts proving that a diminution of blood in the medulla oblongata produces convulsion.

If I have been so particular in quoting every link in the chain of causes and effects in the theory of Marshall Hall, it is because he deserves the great credit of opening a safer path to prosecute our inquiries, placing the first land-mark in this unknown region of pathology. He unravelled the skein of laws regulating the nervous system, and every principle he unfolded proved a mine of new discoveries. With remarkable clearness of sight, he saw that the medulla oblongata, as afterwards confirmed, is the primary seat of epilepsy, and the idea of proclaiming paroxysmal nervous diseases as one and the same disease differing in degree, is, indeed, pregnant with most sound practical applications. Regarding the importance of trachelismus and laryngismus, and of tracheotomy proposed as a preventive security against the effects of the attacks, it is certain, that it was exaggerated by Marshall Hall, for trachelismus and laryngismus prove insufficient to account for the unconsciousness, which is not a consecutive but one of the very initial phenomena with epilepsy; nor do they explain either the asphyxia otherwise occurring many times without occlusion of the glottis. Let it be, however, stated, in justice to the great physiologist, that he did not urge tracheotomy as the specific remedy for every case of epilepsy. An

unbiased perusal of his writings will convince us that his views on the subject are not unfrequently misrepresented. That this is so, I am assured by a personal knowledge of what Marshall Hall did think about tracheotomy shortly before his death; but there is further at the end of one of his latest works the following evidence: "*It is stated that I propose tracheotomy to cure epilepsy, this is to pervert my meaning. I repeat that I only urge this mean to save some patients afflicted with such dire disease in many of its most imminent forms.*"*

Next to Marshall Hall, Brown Séquard is one of the physiologists who has attempted in an indefatigable manner to elucidate the origin of epilepsy. He discovered that a convulsive affection, very much resembling epilepsy, may be produced in animals whose spinal cord has been submitted to certain injuries, and that it is evidently a peculiar kind of irritation starting from the cutaneous ramifications of some centripetal nerves, which alone possesses the power of producing the epileptiform convulsions.

It results from the facts leading to these conclusions:

"1. That the spinal cord in animals may be the *cause* (not the *seat*) of an epileptic affection.

"2. That there is a mysterious relation between certain parts of the spinal cord and remote parts of the skin of the face and neck.

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* Aperçu du Système Spinal, p. 213.

"3. That epileptiform convulsions may be the constant consequence of slight irritations upon certain nerves.

"4. That the trunk of a nerve may not have the power of producing convulsions, whilst its ramifications possess this power.

"5. That even when an epileptic affection has its primitive *cause* in the nervous centres, some cutaneous filaments of nerves, not directly connected with the injured parts of these centres, have a power of producing convulsions, which other nerves, even directly connected with them, have not." *

Then again—"Epilepsy seems to consist especially in an increased reflex excitability of certain parts of the cerebro-spinal axis, and in a loss of the control that, in normal conditions, the will possesses over the reflex faculty. The base of the encephalon, and especially the medulla oblongata, is the most frequent seat of the increase in the reflex excitability, so that this part of the nervous centre is the ordinary seat of epilepsy. The disturbance in the functions of the cerebral lobes, during and immediately after a fit, and in the interparoxysmal periods, is chiefly due to alterations taking place in the brain during the fits. This hitherto mysterious coincidence of loss of consciousness, or, in other words, loss of function of the cerebral lobes, with an increased action of the base of the encephalon, in a complete epileptic seizure, may now

* Physiology of Central Nervous System. Philad., 1860, p. 179.

be easily explained. I have tried to show that the same cause that produces the first convulsions in some muscles of the neck, the eye, the larynx, and the face, produces also a contraction of the blood vessels of the brain proper, which contraction is necessarily followed by the loss of consciousness."* Not in harmony with these statements, Brown Séquard has previously tried to persuade us, that: "We must not, however, conclude from these experiments (those of Todd, Weber, Martin, Magron, and his own) that the seat of epilepsy is only and always in one or in all of these parts—tubercula quadrigemina, the Pons Varolii and the medulla oblongata." And from this, he maintains: "that a change in the reflex excitability of the cerebro-spinal axis chiefly constitutes epilepsy."†

The theories of Brown Séquard and Marshall Hall, are imbued with the same spirit. Yet, Brown Séquard essentially differs from his predecessor in that he holds the seat of epilepsy to be variable, and susceptible of proceeding from an excitation of the whole cerebro-spinal system. He ascribes the unconsciousness to the contraction of the blood-vessels of the brain proper, and accumulation of venous blood charged with carbonic acid, having a special action upon the brain. Finally, he looks upon the fixed state of the chest and not upon the occlusion of the glottis as the efficient cause of asphyxia. If a portion of the views advanced by Marshall Hall are contradicted by some experi-

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* Loc. cit., p. 183. †Researches on Epilepsy, pp. 55–75.

ments of Brown Séquard, these, nevertheless, uphold the fundamental principles set down by the former, whilst the new theory, thus originated, bears not as definite a stamp as that of Marshall Hall; for actually Brown Séquard leaves us in the same incertitude as to the primary seat of epilepsy, in which we were prior to the doctrine of Marshall Hall.

Professor Panum, of Kiel,* has demonstrated that the phenomena of irritation, ascribed by Brown Séquard to the increased production of carbonic acid in the blood, are only due to a want of arterial blood in the great nervous centres — brain, medulla oblongata, and spinal cord. These phenomena of irritation vanish the sooner, the more sudden and complete is the interruption of circulation, and on the contrary persist longer and assume higher proportions, the less complete the interruption; but in either case the supply of blood carried to the nervous centres should be insufficient to entertain life. These transient symptoms of the nervous centres, evidently originate in their structural alteration consequent on local anæmia pushed to the extreme. It is evident that such phenomena would soon lead to death if the nutrition of the nervous substance be abruptly checked upon sudden and complete withdrawal of the circulation; conversely, agony is prolonged if a less sudden and complete stoppage of blood hinders the nutrition with-

* Recherches Experimentales sur les Embolies. Archives Gén. de Médicine. Paris, Sept., 1863, and Archiv. für Pathologische Anatomie und Physiologie, tom. XXV.

out at once destroying the vital qualities of the nervous texture. Dr. Charles Bland Radcliffe has also objected to the views of Brown Séquard, regarding the influence of venous blood to stimulate the irritability in nerve and muscle. He believes: that "the convulsion and the contractions may be more easily explained by referring them to absence of arterial blood, than by referring them to presence of venous blood; for similar convulsions and similar contractions, fact by fact, may be produced when absence of arterial blood is the sole assignable cause. Thus, for example, an animal is not less certainly thrown into a state of general convulsion by hemorrhage than by suffocation, and assuredly it is more philosophical as well as more easy to adopt this latter explanation; for in supposing that venous blood is, as it may well be, the equivalent of absence of arterial blood, a cause is obtained which is of avail, not in one case only but in both cases equally — in the case in which convulsions and contractions are associated with presence of venous blood, and in the case in which convulsions and contractions are associated with absence of arterial blood." *

The hypothesis in question of Brown Séquard equally disagrees with the remark already made by Foville and Van der Kolk; that — "when the fit is at its height and the determination of blood is strongest, the attack oftentimes terminates suddenly, and consciousness

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* Lectures on Epilepsy, Pain, Paralysis, etc. London, 1864, p. 90.

returns, which it would be difficult to reconcile with such theory."*

Finally, I am unable to harmonize such views of Brown Séquard with the following statements made by himself, in a most interesting lecture on the Importance of the Application of Physiology to the Practice of Medicine and Surgery. In speaking of the means of restoring life in syncope, he says: "If you add to that cause of revival (pressing of the sternum and giving a hard push to the heart) another, which is most powerful, and which is directly the reverse of what John Hunter tried upon himself when he found he was in a state of syncope one day at the College of Surgeons; if, instead of making the patient breathe as quickly as he can, you stop his breathing altogether, just as if you were trying to kill him by suffocation, you revive him. By producing a state of asphyxia, for about half a minute, the patient may be saved—he will have a struggle, and come out of it very quickly." †

Although there is a great similarity in the main points of the theories of Marshall Hall and Brown Séquard, the latter, however, took a most important forward step in pointing out the influence of the sympathetic in causing the contraction of the cerebral blood

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*Van der Kolk—On the Minute Structure of the Spinal Cord and Medulla Oblongata, and on Epilepsy. New Sydenham Society, London, 1859, p. 228, and art. Epilepsie, Dict. de Méd. et Chir. Pratiques. Paris, 1831, tom. VII, p. 421.

†Dublin Quarterly Journal of Medical Science, No. LXXVIII, May, 1865, p. 434.

vessels. He, furthermore, manifested with his experiments the peripheral origin of fits, and that, even when the affection has its cause in the nervous centres, the paroxysms may be induced by the irritation of nerves not directly connected with the injured parts. Brown Séquard does not show any reason for this mysterious phenomenon, which presents itself in the relations of these nerves with the oblong medulla.

Such were the immediate effects of the impetus given by Marshall Hall, and the leading facts in the doctrine so ingeniously framed by Brown Sèquard, when Schröeder Van der Kolk, with the lucid results of his microscopic researches confirmed the view, that the medulla oblongata is the original seat of epilepsy. According to this anatomist,* the proximate cause of epilepsy consists in an exalted action of the ganglionic cells on the medulla, which are more or less rapidly exhausted by the spasms and exalted capacity for action. A change takes place in the cells, and it is not until the exhausted irritability is restored by some rest, and fresh nutriment, that the capacity for reflex action once more attains the height at which a slight stimulus, or even no external irritant, is required to excite convulsions. For this reason, again, it is that although the exciting cause, and the more excitable state of the medulla oblongata so produced, are constant, the phenomena are, nevertheless, intermittent. The excited action of the ganglionic cells on the me-

* Op. cit., p. 226.

dulla oblongata must extend its influence to the vaso-motor nerves of the brain, and this altered and more or less disturbed state of the circulation is the cause of the unconsciousness during an attack of epilepsy, while it is incorrect to suppose that this loss of consciousness always precedes the attack. Nor is it a cause of epilepsy, but the result of the altered circulation in the brain, which arises in the beginning of the attack, from the effect of the spasms upon the cerebral vessels. To produce epilepsy, increased excitability, and commonly augmented determination of blood and chemical change are required. It is not necessary to excite this exalted capacity for reflex action on the medulla oblongata, that the stimulus should always be applied through a spinal nerve, the sympathetic and the vagus effect the same.

Careful and special microscopical examination of the medulla oblongata, exhibited an abnormal vascularity with the following alterations, which are also sometimes found in the brain. If the disease has already lasted long, organic vascular dilatation takes place in the medulla, the consequence being that too much blood is supplied, and the ganglionic groups are too strongly irritated, too quickly overcharged. Every attack then becomes a renewed cause of a subsequent attack, as the vascular dilatation is afresh promoted by every fit. Lastly, increased exudation of albumen ensues from the now constantly distended vessels, whose walls at the same time become thickened, producing increased hardness of the medulla,

subsequently passing into fatty degeneration and softening, and rendering the patient incurable.

In epileptics who bite their tongue, the vessels are wider in the origin and course of the hypoglossus, while in those who do not bite the tongue, the vessels in the path of the vagus are wider. With these changes, vascular dilatation in the brain, and particularly in the cortical substance goes hand in hand. The small ganglionic cells, which are here present in such great abundance, become compressed by the dilated vessels, and perhaps in consequence of the more albuminous nature of the intercellular fluid. Dullness and loss of memory are the results; or if after a fit, an unusual current of an arterial blood is supplied, we have, following immediately upon the paroxysm, over-irritation, rage and acute mania, which is present in so many epileptics.

I may notice here, that Van Swieten acknowledges very distinctly the medulla oblongata as the efficient cause of epilepsy. "*Verum illi motus musculares produci nequeunt, nisi causa muscularis motus ab encephalo per nervos veniat ad musculos; adeoque causa epilepticum paroxysmum producens debet efficere, ut medulla encephali in nervorum origines validissimas tantorum motuum causas derivet.*"* These lines seem to have passed unobserved by every writer on epilepsy, excepting Portal, who admits: that, the cerebral cor-

* Commentaria in Hermanni Boerhaave Aphorismos de Cognoscendis et Curandis Morbis. Tomus Tertius. Lugduni Batavorum, MDCCLIII, p. 401.

tical substance and the medulla oblongata, especially, are the efficient or immediate cause of epilepsy, although he judges as rather too exclusive the above views of Van Swieten. And it is no less worthy of mention the following comments of Portal, on a case communicated to him by Ribes. The patient, a soldier, consecutive to a gun-shot wound of the skull became epileptic, and recovered, seven years after infliction of the wound, on natural expulsion of the bullet. In speaking of the lesion sustained by the brain, Portal says: "The dura-mater must have been detached at this site, upon rupture of its cellular tissue and minute vessels, thereon pushing the layers of the arachnoid and pia-mater against the cerebral cortical substance, the convolutional cortical substance has consequently suffered, and the medulla oblongata—*the common centre of our sensations*—has also become disturbed to the point of inducing the epileptic attacks, with more or less congestion of its blood-vessels."* It is, indeed, curious, that after the lapse of over forty years, these assertions, which Portal ventured as a *probable* explanation, should prove to be so perfectly in accordance with our correct knowledge of epilepsy.

The foregoing facts disclosed by Van der Kolk, were the first to evince the organic changes regularly undergone by the nervous centres. And while the eminent anatomist turned the experiments of Bernard,

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* Observations sur le Nature et le Traitement de l'Epilepsie. Paris, 1827, pp. 159 and 187.

Brown Séquard, Schiff, Callenfels, Kussmaul, and others, as evidences of the views he advanced concerning the influence which the excited action of the ganglionic cells has on the vaso-motor nerves of the brain, he also opposed the undeniable results of his microscopical examinations, namely, the material lesions of the medulla oblongata, to the speculations of those who even now persist in contradicting the existence of those lesions, merely because it upsets doctrines rendered untenable by the progress of science. Indeed, Van der Kolk has thrown considerable light on the subject of epilepsy, particularly in what concerns the action of the ganglionic cells, and the intermission in the phenomena they manifest when affected by a persistent irritant; whereas he has no less elucidated the etiology of other convulsive and neuralgic diseases not unfrequently depending on organic changes of the brain and medulla oblongata.

Marshall Hall, Brown Séquard and Van der Kolk explained the origin of epilepsy in conditions related to the more excitable activity of the spinal cord, at the same time they did not disregard the state of the cerebral circulation consequent thereon. It is unnecessary to repeat the views of these authors in reference to the circulatory changes in epilepsy; suffice it to say that, excepting Van der Kolk, they have looked upon them as one of the effects of the epileptic paroxysm. Not so with Van der Kolk, who distinctly establishes—"that to produce epilepsy, an augmented determination of blood and chemical change are re-

quired." Whether the derangement in the nutritive process, necessary to the development of the convulsions, is really induced by a determination of blood, or by a contrary state, is a point to which I shall return; but be as it may, it is unquestionable that Van der Kolk thus clearly points out the efficient part that the blood takes in the causation of epilepsy. And how could it be different, inasmuch as the activity of the spinal cord is entirely dependent on its nutrition, and therefore entertained by the blood? No sooner does a deviation happen from its normal condition, than the functions of the nervous system are thereby deranged. This fundamental principle in the doctrine of Van der Kolk, is the long established aphorism *sanguis moderator nervorum*. The blood provides the organic elements with the constituents for their life, and no systems exhibit greater intimate relation with each other than the nervous and circulatory. Standing on this same position, Russel Reynolds * holds: that "the immediate or proximate cause of convulsions is the same in all instances; it is a change in the nutritive or interstitial processes of the nervous centres."

There is a humoral theory of epilepsy imagined by Todd,† which may at first appear supported by the coincidence of convulsions and blood poisoning. According to it, "the phenomena of the epileptic fit

* Epilepsy, etc. London, 1861, p. 12.

† Clinical Lectures on Paralysis, Certain Diseases of the Brain, and other Affections of the Nervous System — Second edition. London, 1857, p. 299.

depends upon a disturbed state of the nervous force in certain parts of the brain — a morbidly disturbed polarity. This may take place, under the influence of some poison, which may have an affinity for those parts, such as prussic acid, in the same way as strychnine induces an exalted polar state of the spinal cord, or from some disturbance of nutrition, which may be strictly local or sympathetic. This undue exaltation of the polar force induces, subsequently, a state of depression or exhaustion, not only in the parts primarily affected, but in parts of the brain connected with them, according to the degree of the primitive disturbance; just as undue muscular action exhausts the muscular force. The disturbing cause may operate primarily, upon parts of the brain, more directly concerned with the phenomena of consciousness, as the hemispheres; or upon parts, which, when excited, may cause convulsions, as the mesocephale, the region of the tubercula quadrigemina. If the former be chiefly affected, and the latter slightly or not at all, convulsions are either very slight, or do not constitute a part of the epileptic fit. If, on the other hand, the latter are chiefly and primarily disturbed, convulsions form the prominent part of the fit. Now, the exciting cause of all this disturbance generally operates equally on both sides of the brain. But it may operate more on one side than on the other. It leaves behind it a more or less exhausted state of brain; which, again, will be most upon that side upon which there has been the greatest previous excite-

ment. This state of exhaustion is very apt to continue as one of weakened nutrition, in which the brain tissue is more or less in the condition of white softening. If the parts involved in this be the convolutions, mental power, memory, perception, suffer; if deeper parts, as the deeper parts of the white matter of the hemispheres, with the corpora striata and optic thalami, then we have hemiplegic paralysis." Ingenious as this theory is, it fails to be sustained by any evidence, for the existence of such a poison, which gradually accumulates in the blood, and has a special affinity for the brain, is a pure hypothesis.* I could not look at any great length into the speculations brought in support of these views; but could we conceive more numerous causes of production, or more anomalous and capricious incubation of a morbid poison? It is now admitted, that in uræmia and other similar instances of blood poisoning, it is not to the direct excitation of the poison, but to the increased irritability of the spinal cord, and to the greater liability to reflex action, consequent thereon, that convulsions are due. The experiments with strychnia, made by Van der Kolk and other physiologists, afford sufficient evidence to the fact, and it seems strange that Brown Séquard, who, from the beginning, condemned the theory of Todd, should have run so much himself into the humoral doctrine with his hypothesis

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* See Med. Times and Gazette, August 5th, 1854, p. 129, for the supposed generation of this poison, and its way of action.

eoneerning the efficient part which the aecumulated earbonie acid in the blood has in the production of convulsions. It is evident that on the mutual relations of the blood and tissues lies the souree of normal nutrition; but, if the poisoned condition of the blood, whether by earbonic acid or any other agent, were the cause of convulsions and organie ehanges thereby produeed, it is impossible to understand how the fluid circulating throughout the nervous centres, should determine a morbid modifieation only in a redueed small region. It is not, therefore, in the aecumulation of earbonie acid in the blood, whieh is present, again and again, without determining convulsions, but in the peeuliar condition originated in the oblong medulla, that is to be found the initial eause impairing the normal proeess of nutrition, which once disturbed, leaves the organ suseeptible to renewed attaeks. I must lastly admit that Todd, notwithstanding his hypothesis so much devoid of truth as to the cause of epilepsy, did not, however, mistake the seat of the disease, for he fixes it in the mesoeephale and the region of the tubercula quadrigemina, which doubtless are usually involved in epilepsy.

It has been a eommonly aeknowledged opinion, handed down from Hippoerates, that cerebral eongestion is the cause of epilepsy, until Marshall Hall advaneed that the eerebral congestion was secondary to the epileptic seizure, and determined by the spasmodic contraetion of the muscles of the neck — trachelismus and laryngismus. Although Foville rejeeted as in-

correct the idea of cerebral congestion,* showing that the loss of consciousness occurs previous to any congestion, and that the attack frequently terminates, when the paroxysm is at its height, and the determination of blood is strongest, the theory of congestion has, nevertheless, kept many adherents in France. Among the most conspicuous Cazauvieilh, and Bouchet attempted to localize the epileptic disease in a chronic induration of the white cerebral substance, the result of congestion. Bouchet argued subsequently, that congestion was the common essential element of epilepsy, apoplexy, hysteria and mania; and, more recently yet, some writers, among whom stand chiefly Romberg, Van der-Kolk, Reimer and others, admit that plethora and too strong congestion may excite convulsions. However much we might regard the opinion of such authorities it is evident, according to Van der Kolk himself, and to the principal facts adduced on behalf of his theory, that there is no ground to affirm that congestion is the primary cause of epilepsy; for it is demonstrated by the experiments of Bernard, Brown Séquard, Schiff, Donders, Callenfels, Kussmaul and Tenner, and many others, that it does not exist at the inception of the convulsive paroxysms. Such views are again inconsistent with what Van der Kolk says, "that in the very beginning of the attack more or less vascular spasm may exist, which may even produce a momentary retardation or obstruction in the circula-

* Dict. de Méd. et Chir. Pratiques, art. Epilepsie, p. 521.

tion through the capillaries rapidly passing into dilatation, but I should not consider this spasm to be the cause of the attack, but rather the result of the commencing discharge of the nerve ganglia, which are certainly most closely connected with the vaso-motor nerves."* Notwithstanding the exception taken by Van der Kolk, it is no less a fact that cerebral anæmia is among the very initial phenomena of the epileptic paroxysm. Todd establishes with great truth, that: "The vessels of a part, all important as they are to its nutritive and other vital actions, are, nevertheless, only secondary elements in the construction of the organ; and unless in themselves diseased, they can play only a secondary part in the production of organic or functional derangement. Congestion of blood vessels, or hyperæmia of a part, must be an *effect* either of some disordered state of the intrinsic elements of the tissue, or of the blood, or of the forces by which the blood circulates. And a sound pathology ought to receive no other explanation of morbid phenomena, or of congestion, if it exist, but that which traces the real state of these."† And here, let me further quote, from a clinical lecture on Apoplectiform Congestion and its relation to Epilepsy and Eclampsia, the following words of Trousseau, which bear strongly on this subject: "There are, therefore, and I lay great stress on the point, two very distinct conditions in an attack of eclampsia, or of epilepsy, whether idiopathic or

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* Op. cit., p. 298. † Op. cit., p. 299.

symptomatic. 1st. A *cerebro-spinal modification*, unknown in its essence and in its nature, which in a second abolishes all the manifestations of animal life. Of the two this is by far the more important condition. 2d. A *secondary cerebral congestion*, which although less important, may in some extremely rare cases be carried so far as to produce subcutaneous ecchymoses, cerebral capillary hemorrhage, and even meningeal hemorrhage."*

The more recent and very valuable physiological and pathological researches on cerebral softening made by Provost and Cotard, have added stronger evidences against the theory of cerebral congestion as cause of epilepsy. These authors have found that if dust of lycopodium suspended in water is injected into the carotid artery "immediately after the injection, the animal generally utters a moaning, is seized with convulsions, and dies exhibiting the same symptoms, as on the case of ligature." If a powder not as minute as the sporules of lycopodium be employed, and that consequently the circulatory system is not so generally obstructed, convulsion may not occur.

Passing from the physiological experiments to the pathological cases reported by Provost and Cotard, we find that in five cases in which epileptiform convulsions existed along with the cerebral softening, the accident could be traced to the four following causes :

* Lectures on Clinical Medicine, translated with notes and appendices by P. Victor Bazire, M. D. London, 1866, p. 36.

1st, Thrombosis of a cerebral artery. 2d, Capillary embolism. 3d, Atheromatous state of the cerebral arteries. 4th, Laceration of the cortical substance from hemorrhagic softening, a fact frequently noticed by Charcot coinciding with epileptiform convulsions.* I shall have occasion hereafter to describe cases presenting pathological changes of the brain similar to those referred to by Provost and Cotard. Finally, let me add, that no higher degree of cerebral hyperæmia could be than that found in those dying during the algid stage of cholera, and yet, no unconsciousness, nor impairment of mind, nor convulsions are observed with it, which evidently proves: that something more than the congestion, or rather different from it, is necessary to originate epilepsy.

Little did Henle† succeed in his attempt to establish that epilepsy was due to a pressure by accumulated blood in the vessels of the brain. Admitting two forms of epilepsy, one with plethora and another with anæmia, and assuming that the quantity of blood remains unchanged in the cerebro-spinal cavity, Henle supposed: that in anæmia the blood from the upper part of the encephalon necessarily fills the blood vessels of the base, thus causing unconsciousness, which in plethora is determined by the general compression of the blood in the brain proper. This hypothesis,

* Etudes Physiologiques et Pathologiques sur le Ramollissement Cérébral. Paris, 1866, pp. 6 and 131.

† Handbuch der Rationale Pathologie. Vol. II, 1855, part I, p. 181, and part II, p. 46.

resting on no facts whatever, met with no favor. Not so, however, with another very important theory, also German, of Kussmaul and Tenner, ascribing the proximate cause of epilepsy to sudden cerebral anæmia.

The hypothesis that epilepsy may depend upon cerebral plethora was fully contradicted for the first time by the interesting experiments of Kussmaul and Tenner. They observed, on repeated occasions, "that section of the cervical sympathetic and ligature of the jugular veins was followed by protrusion of the eyeballs, slower respiration, paralysis of the glottis, weakness in the legs, without, however, loss of consciousness, and only with slight transient convulsions." The same physiologists have, on the contrary, proved, "that sudden arterial anæmia of the brain, as also faradization of the cervical sympathetic nerves, which determines permanent spasms of the blood vessels gives rise to epileptic fits. Nutrition is thus suddenly impeded, and the brain brought into that peculiar internal condition which is manifested in the form of an epileptic attack. Epileptic convulsions are likewise induced when the arterial blood rapidly assumes a venous character, and it is highly probable that the spasms depend upon the suddenly interrupted nutrition of the brain. Epileptic convulsions in hemorrhage do not proceed from the spinal cord, neither from the cerebrum, their central seat is to be sought for in the excitable districts of the brain lying behind the thalami optici. Anæmia of those parts of the brain

situated before the crura cerebri produce unconsciousness, insensibility, and paralysis in human beings; if spasms occur with these symptoms, some excitable parts behind the thalami optici must have likewise undergone some change. Anæmia of the spinal cord produces paralysis of the limbs, of the muscles of the trunk, and of respiration. When the anæmia suddenly attains its greater intensity, then only and even then but rarely, do slight trembling movements of the limbs precede paralysis. Arterial congestion of the brain does not seem to be capable of producing any other symptoms than those of paralysis (dizziness and apoplexy). Venous congestion of the brain, as well as arterio-venous congestion, brings about conditions which belong more to those of apoplexy than to those of epilepsy, and are characterized by paralysis of the glottis, together with a slower respiration and slight spasmodic symptoms. All theories are false which assert the epileptic attack to be derived from a sudden determination of blood, whether active, passive or mixed. It is probable that certain forms of epilepsy result from a spasm of the muscular coats of the cerebral arteries. The epileptic affection, which disposes to the attacks, occupies either the whole of the brain, or some districts only, and by it the brain is brought into that altered state on which the epileptic attack is based. The medulla oblongata, as being the part whence the nerves causing the constriction of the glottis, and the vaso-motor nerves take their rise, seems frequently to be the spot from which eclamptic

and epileptic attacks proceed. Circumscribed anatomical alterations of the brain, or alterations of protracted duration cannot be regarded as the proximate cause of epileptic attacks, but may cause epileptic affections (dispose to epilepsy). Pathological anatomy cannot give any explanation as to the nature of epilepsy." *

The researches of Kussmaul and Tenner refer to one element only, in the pathogeny of epilepsy. Their experiments, in addition to those of other physiologists, establish the chief share which the sympathetic has on the determination of spasms, and confirms the correctness of the statements on the subject advanced by Brown Séquard and Van der Kolk. They peremptorily show how physiological experiments and pathological investigations agree with each other. Such experiments are, in fact, the complement of what Marshall Hall and Van der Kolk, had emphatically asserted about the seat of epilepsy, for they prove that the *nodus epilepticus* is in the medulla oblongata, and these very facts furthermore confirm that there is a material modification in every case of epilepsy. Nevertheless, there is one point in the conclusions of Kussmaul and Tenner, to which I except, namely, "that pathological anatomy cannot give any explanation as to the nature of epilepsy." The origin of epilepsy is certainly not in the conditions of the cere-

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* On the Nature and Origin of Epileptiform Convulsions, etc. New Sydenham Society, London, 1859, pp. 105, *et seq.*

bral circulation, nor in the state of the blood, neither in the brain, nor the peripheral lesions, nor the different organs which may be induced into convulsions, but in the very modification undergone by the medulla, or to be more precise, in the over-excited action of its ganglionic cells controlling the vaso-motor elements and nutrition. The paroxysm may well occur without muscular spasms; it does not, however, occur without a disturbance of the vaso-motor elements in its inception, and hence: paleness of the face, or loss of consciousness, or vertigo, not unfrequently are the whole symptoms of the epileptic attacks in the early stage of the disease. I deem it conclusive, that in this over-action of the reflex centre, and the influence it exerts on the vaso-motor nerves, and in no other condition, is to be discovered the true explanation of epilepsy. That such modification is readily attended with a definite change in the vegetative process of the nervous structure, is most certainly warranted by the anatomo-pathological researches of Van der Kolk, and those I am about to relate. Axenfeld says in his valuable work on Neuroses* — "Epilepsy may attend a slight lesion, such as partial ossification of the falx cerebri, whereas it may again be absent with a deeper disorganization as that in extensive softening of the brain; it may supervene on the occasion of any cause, still capable of being present without giving rise

* Elements de Pathologie Médicale, par Requin, tom. IV, Paris, 1863, p. 572.

to it, while it will often exist in a grave and long-standing form and, yet, no change be disclosed by the examination of the encephalon, at least nothing discoverable by our actual means of investigation. Must we thereby conclude that, when met with, the organic modifications of the nervous centres are mere coincidences? Most certainly not; what must, however, be acknowledged, is, that between the lesion and the symptom, epilepsy, there lies an immediate condition, a less tangible element, the only necessary one, always invariable in the midst of such a diversity of apparent conditions, and capable of being realized when every other may be wanting." This invariable and necessary element, is, I will repeat it, the over-excited condition of the ganglionic cells in the medulla oblongata. The following experiments of Brown Séquard cannot escape attention, as they bring further proof to these statements. "If we take two guinea-pigs, one not having been submitted to any injury of the spinal cord, and the other having had this organ injured, we find, in preventing them from breathing for two minutes, that convulsions come in both; but if we allow them to breathe again, the first one recovers almost at once, while the second continues to have violent convulsions for two or three minutes, and sometimes more." * Leaving aside the true nature of the influence which accumulated carbonic acid in the blood might exert in stimulating the action of the cerebro-

* Researches on Epilepsy, p. 5.

spinal axis,* it is evident, that the reason why the second animal continues with violent convulsions, is only in the increased impressibility of the injured spinal cord, which also increases its readiness of action. Evidence of this fact is again evinced in the liability to epilepsy exhibited by persons returned to life after they had attempted suicide by hanging. There is, beside, a most remarkable experiment, made by Vulpian, which conspicuously shows, not only that the seat of epilepsy is not in the brain, but also that withheld circulation does not determine convulsions unless the ischemia extends to the oblong medulla. "I have seen," says Vulpian, "in a rabbit, the compression of the carotid and vertebral arteries, determine a suspension of the cerebral functions; but a thing most remarkable! spontaneous respiration continued, the medulla oblongata having more or less completely escaped from cerebral anæmia. Spontaneous and reflex movements had totally disappeared in the face and in the eyes, while the trunk of the animal yet lived sustaining a head physiologically dead. Now, in the course of two or three minutes, the means of compression being removed, convulsive movements took place though lasting a very short time — afterwards, all vital manifestations, all voluntary and other movements gradually returned in the head, the animal recommenced to walk and soon recovered his normal state." †

* Journal de la Physiologie. Paris, 1858. tom. 1, p. 95.

† Leçons sur la Physiologie du Système Nerveux. Paris, 1866, p. 455. This experiment also manifests, that the accumulation of

In a course of lectures delivered in 1864 before the College of Physicians in London, Dr. Charles Bland Radcliffe endeavored to enforce the theory which he had already advanced twelve years before, to introduce a radical change in our current knowledge of muscular contraction. In this theory Dr. Radcliffe asserts: that muscular contraction is a purely physical process, no muscular tonicity or irritability, or natural electricity converted into contractile force, being concerned in bringing the muscle into the state of contraction. This is associated with deprivation of nervous force and of natural electricity, and not with a contrary state of things, the ordinary muscular contraction and rigor mortis being, therefore, only different aspects of the same process. These and other physiological considerations lead to the general conclusion, that: convulsion is connected with a state of depressed vital energy, in which the functions of respiration, circulation and innervation are consequently in a state of depressed or exhausted action.* Dr. Radcliffe has tried, with a great deal of research and remarkable ability, to prove the correctness of his views by evidence and experiments borrowed from our leading physiologists, as well as with many original arguments and observations. Assuredly, the doctrine has points which need greater distinctness to be unreservedly accepted, although it broadly reflects light over many obscure nervous dis-

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blood charged with carbonic acid may take place in the brain, without superinducing convulsions.

* Op. cit., Lectures IV, and V.

orders, and the arguments borne out by the new physiology of muscular motion conduct its learned author to sound conclusions concerning the pathology and therapeutics of epilepsy, and worthy of the most serious consideration.

It is impossible to pass in silence the views expressed by Jules Falret, one of the most eminent mental pathologists in France.* He objects to considering the oblong medulla as the principal seat of epilepsy, or to tracing to it the reflex origin of the disease, and with Georget, Calmeil, Herpin, and others, denies the epileptic *aura* and the influence of peripheral excitation in determining the spasms. After thus setting aside the physiological theories, already reviewed, Falret establishes, that: "the essential seat of epilepsy is in the brain, *i. e.*: in that part of the encephalon presiding over the manifestations of our mind, sensibility and voluntary movements, and not in that which is the centre of reflex or excitomotory power." The facts upon which these adverse conclusions are based do not give them any real countenance, inasmuch as the phenomena they are intended to clear up are more satisfactorily interpreted and explained by the positive experience of those whose theories are so completely rejected by Falret. The days are gone by in which science was ruled by the most plausible hypotheses; notwithstanding his judicious and progressive mind, Falret encourages the belief of those who look yet upon

* Arch. Gén. de Méd. Paris, Juillet, 1859.

the endeavors of experimental physiologists to elucidate our knowledge of nervous diseases as vain attempts. The words below, of a talented and philosophical writer, Guardia,* should be addressed to those who condemn the onward movement of science, because they chose not to listen to the rational explanations of disease by the discoveries of modern physiology. "Since the works of Bichat pathology and physiology are acknowledged independent of each other, disease being not an essence, or entity, or abstraction, but simply an abnormal manifestation having its cause and seat in the organic elements, without which there are no vital properties. Now, the different manifestations constituting the phenomena or morbid states are only symptoms of such modified properties. Medicine, therefore, is truly physiological, and these two words together represent, not the motto of a school or medical sect, but a truth, a doctrine which progressed from Bichat to Broussais, and remains standing in spite of passionate declamations of a senseless reaction."

Having thus come to a close, there naturally occurs the question — what is epilepsy? Were I to present a definition embracing the principal features of the disease, I should say: that epilepsy is a disease constituted by chronic paroxysms, excited upon a direct or reflex action of the medulla oblongata, in a condition of exalted irritability, coincident with sudden depression in the cerebral circulation and with loss of

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* De l'Etude de la Folie. Paris, 1860, p. 20.

consciousness, with or without muscular spasms. This definition does not differ from that of Van der Kolk, which seems to be the most rational. But, it prominently takes into account the unconsciousness; for this is a pathognomonic symptom, oftentime constituting the epileptic paroxysm by itself. I could not admit that the seat of epilepsy is variable, as held by Brown Séquard, because although the spasms may be the result of an excitation of the whole cerebro spinal axis, yet, it is no less evident, as proved by experiments of Brown Séquard himself, that the capacity for reflex movements does not show itself until long after the infliction of the injury to the spinal cord (three weeks). At this time, as justly remarked by Van der Kolk, the injury has extended to the medulla oblongata, which more than any other region of the nervous centres appears endowed with an intermission of activity to discharge its force. Moreover, the fits in the animals experimented upon by Brown Séquard, were only induced by irritating the medulla through the trigeminus and probably the accessory.* These views stand yet on more positive ground, if we further take into account that the phenomena have occurred with more intensity, when the vital knot has been injured, as in the epileptic guinea-pig thus operated upon by Brown Séquard. For all these reasons, and always based on the physiological experiments already quoted, and on the anatomo-pathological researches of Van der Kolk

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* Researches on Epilepsy, p. 6, and Van der Kolk, op. cit., p. 219.

and my own, I am led to admit: that the medulla oblongata is the original seat of epilepsy, and that the disease primarily involves the vaso-motor nerves.

I must refrain from pursuing at present the particulars of further evidence, for sufficient has hitherto been adduced exclusive of that to be necessarily brought out in the following pages. It would be far beyond my design if I were to engage myself in such consideration: I have wished to exhibit in a concise general manner the views I entertain, after having briefly alluded to the characteristics of our principal modern doctrines of epilepsy, which are subjects of deeper and useful thoughts.

CHAPTER II.

PATHOLOGICAL ANATOMY OF EPILEPSY.

The organic lesions of epilepsy have to be sought for in the centres as well as in the peripheral parts of the nervous system. Usually, it is with long standing cases that the derangement becomes so general. There are, however, regions which seem to be the primary seat and never to escape the influence of the disease, of which they display microscopical evidences, even when other parts of the nervous system remain unhurt. These regions, as first demonstrated by Van der Kolk are in the medulla oblongata.

The assertions to be made here are derived from personal experience of twenty-six autopsies of epileptics, most of whom under my charge at the Hospital for Epileptics and Paralytics, or at the Charity Hospital, Blackwell's Island, N. Y. The cause and history of the disease were in some instances unknown, owing to the admission of the patients into the institutions without any record of their symptoms; in every case, however, I have satisfied myself that the disease was genuine epilepsy.

It may be well to state, before going any further, the cause of these twenty-six cases, thus divided:

Unknown in -	11 cases.	Teething in - -	1 case.
Intemperance in -	4 "	Fright in - -	1 "
Syphilis in -	3 "	Scarlatina in - -	1 "
Congenital in -	3 "	Attempted strangulation in - -	1 "
Dysentery in -	1 case.		

The respective sex of these twenty-six patients was: eleven males and fifteen females. Two males and one female of the three congenital cases were also idiotic. The fits were accompanied with dementia in one male and in four females. As it would be too long and tedious to detail every example, I will give an account solely of those manifesting the most important metamorphoses undergone by the nervous system, before presenting a synoptic table with the general results of all the post-mortem examinations.

To proceed in a systematic manner I will describe successively the alterations discovered in: the cranium, the brain and cerebellum, the medulla oblongata and spinal cord, the sympathetic ganglia and peripheral nerves, the circulatory system and the blood.

I have ascertained myself the condition of the skull in sixteen out of the above twenty-six cases. In six, three males and three females, the skull was evidently thicker than normally throughout its circumference. In two males the bones were very thin, in one with syphilitic epilepsy, the tissues had mostly disappeared from a large space of the inner table of the calvarium, leaving a rugged surface and reducing considerably the width of the cranial wall. In another female, there was complete absence of diploë, rendering the bones more dense, but much thinner. Marked deformity of the skull existed only once in a male. I may add, that in the case of a very distinguished physician in Albany, whom I saw in con-

sultation with Dr. Thomas Hun, and who died from epilepsy, the skull, very thin throughout, was reduced to a transparent table along the saggital suture, and particularly near the parietal foramen. The fits occurred within the short period of three weeks: a large abscess with thick vascular walls existed in the anterior part of the brain; the patient did not complain of any prior symptoms excepting headache, with supraorbital neuralgia and vomiting, and the intellectual faculties remained unimpaired to the very moment of the final attack.

The cranial measurement in ten of these sixteen cases shows the following results:

With one female, aged 28: circumference 22 inches. Transverse diameter 13.8 inches. Antero posterior diameter 12.9 inches.

The remaining nine males gave in an average: circumference 21.3 inches. Transverse diameter 13.6 inches. Antero posterior diameter 13.2 inches.

In one of these males, epileptic since infancy and idiotic, the skull measured; circumference 20 inches, transverse diameter 11 inches, antero posterior diameter 10¾ inches.

Comparing these with the results of other measurements, I keep, of 135 epileptics — 80 males and 55 females — I find that the average has been, with the males: circumference 22.2 inches; from one to another auditory foramen passing over the sinciput 13.5 inches; from the root of the nose to the occipital protuberance 13.8 inches.

With the females; circumference 21.8 inches; from one to another auditory forumen, 13 inches; from the root of the nose to the occipital protuberance 13.2 inches.

These results do not differ much from those obtained by Dr. Boyd, of the Somerset County Lunatic Asylum, and quoted by Sieveking in his valuable work on Epilepsy.*

I have twice examined microscopically the structure of the thinned bones of the skull. Excepting an absence of nucleated plates, *myeloplaxes*, and a great diminution in the quantity and calibre of the capillary vessels, I have not been able to detect any special alteration of the osseous tissue, examined in a fresh condition. The more remarkable change I have noticed in relation to the bones of the skull was of a local nature as it only referred to dried fragments of an exostosis removed from the occipital. The inner and outer tables were unusually thickened, and the diploë had a highly cancellated structure, with extremely enlarged cylindrical cavities to lodge the capillary vessels. The tissue of the tables, close textured, displayed very few irregular cavities and some orifices of the vascular canals. The most solid parts exhibited neither Haversian canals, nor lacunæ, and these latter, of irregular form, did not concentrically surround the canals in the less dense structure close to the diploic tissue.

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* On Epilepsy and Epileptic Seizures. London, 1861, p. 168.

In regard to the osseous growths in the dura-mater and the meninges, I once examined two plates situated at the anterior part of the spinal lumbar region of an epileptic child. The inner surface of the dura-mater, of a red tint, united to them by loose yellowish exudations and attenuate capillary vessels. These plates were opaque, the lower one not larger than a pea, the upper forming an irregular oblong patch, about an inch in size, immediately connected with the arachnoid, and projecting on the spinal cord from which it easily detached. Under the microscope neither manifested, however, an osseous or cartilaginous structure. The smaller, white, and cartilaginous-like, was compounded of an amorphous matter, mixed with fatty and calcareous granulations inclosed in the connective tissue of the membrane. The calcareous element predominated in the larger layer, and hence its firmer chalky appearance.

I have never detected any osseous structure in the small patches and nodulated bodies of the cerebral arachnoid, occasionally present after long standing epilepsy. They have been located, like miliary tubercle, near the Sylvian fissure, at the base of the brain, and upon the cerebellum in the form of scales or patches. On three occasions I have met with these deposits in the ependyma of the ventricles and the choroid plexus, thickly covered with granulations of different sizes, such as it has been also often observed by Dr. S. Wilks * in

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* Clinical Notes on Atrophy of the Brain. Journal of Mental Sciences. October, 1864.

cases of epilepsy. The structure of these granulations resembles that just described in the spinal meninges, with an addition of amyloid corpuscles; or they may become altogether fatty as in Case VI. They may beside grow on the inner face of the dura-mater, or as a very thin film on the surface of the arachnoid with which they are firmly soldered without having any connection with the brain. The only true ossification I have discovered, existed in the dura-mater along the posterior part of the falx cerebri, about one inch from its upper margin, and not far from the tentorium cerebelli. The osteophyte, removed from an idiotic epileptic, was surrounded by old apoplectic effusions in the cortical substance of the brain. The plate was about an inch in surface, whitish, not very and irregularly thick, and without any attachment to the arachnoid. But though some portions of the adventitious growth exhibited the characters of genuine bone, their minute structure was quite imperfect, with very rare bone corpuscles, and a few canaliculi irregularly situated in the midst of a calcareous looking substance, with no medullary elements. The rest of the peripheral tissue of the plate, was composed of elastic fibres, easily disintegrated, and filled with fine calcareous and fatty granulations. This and the preceding specimen were brought to me by Dr. Lyman, Resident Physician at Bellevue Hospital.

The dura-mater was the seat of extensive ulceration in Case V. The sinuses have been plugged or completely obstructed by a non-inflammatory process, as it happened

in the same case, Plate II. In this instance the whole longitudinal sinus had been replaced by a hard white fibrous tissue. Further particulars of this interesting specimen will be given with the report of the case, p. 68, which was one of syphilitic epilepsy. Thrombosis consequent on non-inflammatory process, in the majority of instances, takes places in the longitudinal sinus. I am aware of one case, only, in which Gerhart found the straight sinus affected.* I can add another instance of the kind, in an epileptic woman, of intemperate habits, who died at the hospital with double pneumonia. Autopsy: meninges and brain bloodless. Straight sinus, thick and hard, filled by a discolored clot, extending into the inferior longitudinal sinus and venæ Galeni, and completely obstructing the straight sinus. Heart fatty, mitral and tricuspid valves thickened. Lungs; pleuræ adherent, pleuro-pneumonia with preponderance of the pneumonic element in the two lower thirds of right lung, and condensation passing into pus on the base of the left. Liver and kidneys fatty. Brain and medulla with the fatty granular degeneration proper to chronic alcoholismus.

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*Deutsche Klinik, 1857, No. 45, and On Thrombosis of the Cerebral Sinus, by Th. Van Dusch. New Sydenham Society, London, 1861, p. 113.

CASE I. *Alternate hemiplegia. Heart disease. Fatty tumor of dura-mater. Aneurism of right middle cerebral artery. Sclerosis of Pons Varolii. Degeneration of the brain, cerebellum and medulla oblongata.* (From notes by Dr. F. A. Castle).

Sarah A. . . , aged twelve, native of New York, entered the Hospital for Epilepties in the morning of January 10, 1867, and died after a paroxysm, the following day in the afternoon. She had been subject to fits, and had paralysis of the right side of the face, and of the left limbs, principally the arm. No history obtained of her trouble.

Autopsy on the 12th, at 10 o'clock, A. M. Thermometer, at 20° Fahrenheit. Body not emaciated, well marked cadaveric rigidity. Eschar under right side of the chin. Cicatrix in right groin. Hypostatic congestion about the back and nates. Chest; extensive old pleuritic adhesions on right side, lower lobe of right lung emphysematous. Heart enlarged. Aortic and mitral valves with fibrinous deposits attached to them. Intestines not examined. Liver, spleen and kidneys normal. Uterus nulliparous. Orifice of urethra swollen and ulcerated. Scalp loosely adherent. Skull of moderate thickness, diploë nearly absent. Membranes easily drawn off. Small tumor the size of a hazel nut attached to dura-mater, on left side, just above the posterior extremity of the lateral sinus. Corresponding depression in the left cerebral lobe underneath the tumor. Arachnoid on right side dark colored, and filled with about two and a half ounces of thick grumous blood and clots. Cerebral convolutions flattened and infiltrated with blood. Such an extravasation proceeded from the bursting of an aneurismal dilatation of the middle cerebral artery near the place where it gives off the anterior branch. Medulla oblongata removed and kept for microscopical investigation.

Different sections of the brain exhibited a punctiform injection throughout, and superficial softening of the cortical substance on right side. Here the cells and nerve tubes were broken up, with great admixture of decomposed blood, and granule cells. No degeneration of any account in the capillary vessels. The occlusion of the middle cerebral artery was complete; its over-distended walls exhibited no structural lesion, and the aneurism was originated by embolism, undoubtedly in connection with the cardiac disease, a mode of formation first pointed out by Dr. John W. Ogle, and substantiated by this and other cases to be here detailed.

The tumor in the dura-mater, not adhering to the meninges, had, as already noticed, produced a depression in the brain. This growth was not the consequence of some old effusion or false membrane, for the tissue in the vicinity had maintained its normal structure without any increased vascularity; neither was it a degeneration of a Pachionian gland, being merely constituted by large fatty cells and granulations, mixed with delicate cellular fibres, contained in a covering of thick connective tissue and elastic fibres. The blood vessels were wanting in this structure.

In the cerebellum, the corpus dentatum of both hemispheres had undergone a granular degeneration, identical with that discovered in the spinal cord. The capillaries in the oblong medulla, distended and embedded in a firm amorphous matter, mostly appeared so in the nuclei of the hypoglossi and along the path of the right pneumogastric. The nerve cells were disintegrated, or loaded with granulations and pigment completely masking their nucleus. The anterior pyramids had a grayish color, due to an increased quantity of neuroglia. This hyperplasia of connective fibres, preponderant at the central lower part of the Pons Varolii and right pyramid, extended along the inner region of the anterior columns, and the anterior cornu on the left side of the spinal cord, which was removed as low down as the fourth cervical nerves. A tranverse section of the medulla at the posterior border of the Pons Varolii, showed the large cells in the nucleus of the right facial very much altered, from either partial destruction or complete disintegration; in some places they were entirely replaced by brilliant molecules of fat, also collected in groups along the path of the nerve. The nerve fibres in the left anterior, and lateral columns were resolved into granules, and the fibres of the anterior commissure in many places presented dark clusters of fatty particles, located more to the left than to the right side. The left tractus intermedio-lateralis, was softer and more friable than the right, particularly opposite the roots of the third and fourth cervical nerves. A number of corpora amylacea were present, though not uniformly, around the spinal canal, and intermingled with delicate transparent fibres of neuroglia; moreover, the connective tissue, very much increased, was likewise thickly interspersed throughout the white columns. The gray substance in its anterior regions was very much softened, frequently traversed by an irregular fine network

formed by the connective tissue, or reduced to a very transparent granular amorphous matter: nerve cells in identical granular condition to those of the medulla, and irregularly shaped. Spinal capillaries distended and thickly covered by exudation. This degeneration seemed more advanced nearer the surface of the anterior and lateral left columns, altering very much the shape of the gray substance, which on section exhibited a dark pellucid appearance. The condition of the columns in the opposite half was not entirely healthy; they appeared in some sections of the cord partially and not so sharply touched, with the connective tissue evidently hypertrophied. The damage was still less marked with the posterior columns, excepting that here the nerve fibres near the cornua were partially destroyed and diffused throughout the more than naturally abundant neuroglia; a change which could be followed upwards into the corpora restiformia. The anterior roots of the nerves in the left side were more or less encroached upon by the degeneration, most of the primitive fibres deprived of myeline, with a number of nuclei in their sheath, appeared translucent and gray. The structure of the cord seemed healthy under a low power, and the above degeneration was distinct with magnifying powers of two hundred and fifty, and five hundred diameters.

The changes already described suffice to account for the alternate hemiplegia of the patient. Owing to the immediate occurrence of death it was impossible to ascertain the state of sensibility in the paralyzed extremities, or if there existed any functional impairment of the eye and chest, or on the left limbs. Equally interesting would it have been to know positively if the girl had any syphilitic disease, as appearances were very suspicious.

The so-called neo-membranes or false membranes of the coverings of the brain have been the subject of interesting researches by several authors, among

whom stand conspicuously: Baillarger, Calmeil, Cruveilhier, Robin, Charcot, Vulpian and Lanceraux, in France, Heschel, Virchow, Rokitansky, Hasse and others in Germany, and Hewitt and Ogle in England. It has been shown by Virchow, that the origin of such adventitious products is a pachymeningitis or inflammation of the dura-mater. They are coincident with a determination of blood in the surface of the cranial membrane, and may become the seat of meningeal hemorrhage. I have met with instances of general paralysis and other cerebral diseases attended by such false membranes, which I have found only three times accompanying epilepsy. Twice they were connected with the dura-mater, and once with the arachnoid. My views as to the inflammatory nature of these adventitious growths, agree with those of Virchow, Ogle and Lanceraux.

The two examples which I will presently allude to, were patients in my division at the Charity Hospital; both inveterate cases of epilepsy, the result of intemperance, and old inmates of the Alms House, without records of their disease.

CASE II. *Epilepsy. Left hemiplegia. Death upon a succession of fits. Neo-membrane of dura-mater. Granulations upon the cerebellum. Degeneration of the brain and medulla oblongata. Fatty heart. Tuberculosis of the lungs.*

Male, aged 54. Intemperate. Epileptic with paralysis of the left limbs. Was seized with severe fits during the night, followed by cerebral symptoms, delirium, coma and death.

Autopsy.—Heart flabby and fatty, as also the liver and kidneys. Tubercuious deposits at the apex of both lungs. Calvarium not ad-

herent to the dura-mater. Yellowish patch on the right side, firmly attached to the inner surface of the dura-mater: it extended from the middle fossa in the base of the brain to the longitudinal sinus, and from the lateral one to the frontal suture. This layer, thicker and firmer over the fissure of Sylvius, very slightly adhered to the arachnoid beneath from which it was without effort stripped off. Effusion of subarachnoid fluid, and extensive fatty degeneration of the right corpus striatum and optic thalamus, and of the cortical substance. The middle cerebral arteries and those of the choroid plexus had undergone an atheromatous change. Cerebral ventricles distended with a large quantity of a whitish serosity. Nothing particular noticed in the cerebellum, excepting the granulations in its covering membranes. The medulla oblongata and the cord were very much congested: the former deeply altered in its structure.

CASE III. *Epilepsy. Right hemiplegia with contraction of the limbs. Death in a fit. Degeneration of the brain, and of the second and third left frontal convolutions and Insula of Reil. Plugging of right vena Galeni, with bursting of right vena corporis striati. Yellow softening of the cerebellum. Lesion of the medulla. No aphasia. Cardiac disease.*

Kate O....., aged 40, of very intemperate habits, with unilateral epilepsy and paralysis and contraction of the right limbs. Had several fits before becoming unconscious, and dying in one paroxysm. Never exhibited any impediment in her speech, on the different occasions that I saw her at the Alms House.

Autopsy.— Heart soft and dilated, aortic valves, thick and atheromatous, the atheroma extending to the arch of the aorta. Lungs congested; lower lobe of the right hepatized. Liver contracted and granular. Kidneys fatty. Calvarium thickened. Dura-mater highly vascular, with false membrane in the inner surface, corresponding to the left middle fossa in the base of the brain. Considerable meningitic effusion and congestion extending throughout to the spinal cord. Brain on section wet, with minute points of redness from capillary congestion. Right corpus striatum, central posterior part of the hemispheres, and gray substance in the convolutions, with evidences of old apoplectic effusions, very conspicuous in several small points of the Insula and of the second and third left frontal

convolutions. In the corpus striatum, the apoplectic cicatrix had a lilac color bounded by a deep yellow dense tissue. A little cavity, not larger than a pea, circumscribed by similar yellow tissue was present in the left cerebral hemisphere. The ecchymotic points in the convolutional gray substance, had a rosy hue. Ventricles considerably distended with serosity and a large quantity of clotted blood, proceeding from a rent in the right vena corporis striati. Septum lucidum lacerated, and veins in the choroid plexus very much dilated; such impediment to circulation arising from plugging of the right vena Galeni. Yellow softening of the cerebellum and of the medulla preponderant in the vagal nuclei.

The false membrane in this instance had a higher organization than in the former. Its smooth inner surface, of pellucid aspect, showed minute capillaries, some of them continued with those of the dura-mater. This surface had also dark purple stains produced by sanguineous exudations. The blood so effused was between the layers of the membrane, which could be readily separated from the dura-mater, and had no connection whatever with the arachnoid.

Microscopical examination manifested the identical texture of the false membrane in either case. The under surface of the dura-mater was increased in vascularity, and covered by an amorphous transparent matter, mixed with fine granulations and nuclei in those regions where the layer, half transparent and of uncertain thickness, appeared in its early stage of growth. Such a condition was particularly noticeable in the former case. At a more advanced period, as with the woman, when the neo-membrane had a complete structure, the connective elements, in excess and in wavy bundles of opaque fibres, were mixed with elastic slender transparent filaments, mingled with brilliant fatty molecules. On the free surface of the layer

there were flattened cells with an oval nucleus, and the characters of lymph cells running towards the epithelial form. The capillaries had a large diameter; they were easily torn, and formed by an inner tunic of perpendicular fibres, with an oval nuclei, whereas the external had transverse fusiform fibres with elongated narrow pale nuclei. The blood decomposed in the stains of the membrane, presented withered globules in a granular state, fine fatty granulations, with crystals of hæmatoidine and granules of hæmatosine.

In another female epileptic, who died at the Hospital for Epileptics, the 11th of March, 1867, the record of the autopsy, made by Dr. McDonal McClung, says; Along the left fissure of Sylvius, in the cavity of the arachnoid, was a thin layer, at first looking purulent, and which proved to be a fibrinous false membrane, developed in this site, without attachment to the brain. This latter exhibited capillary congestion and softening of the white substance in the right hemisphere.

The degenerations of the brain and cerebellum are not primitive with idiopathic epilepsy. I will not refer here to cases attended from their inception with epileptic convulsions, though in them the cause of the correspondence of the convulsions and the cerebral trouble is manifest enough. The changes undergone by the brain are but accidents in the course of epilepsy, the primary lesion of the disease being in the medulla oblongata. It is in this place where the morbid process, which causes the convulsions, breaks forth to reach distant parts. I have met with

instances of epilepsy wherein the brain was free from injury, but I have not yet come across any in which the medulla did not exhibit evident proofs of damage, the microscopical examination having been most careful in every case. If I compare more closely the results of my researches, I find that the regions of the encephalon apt to be the first encroached upon by the disease, are: the floor of the third ventricle, the corpora striata, optic thalami, convolutional gray substance, and peduncles of the cerebellum. I have named these parts in the order of frequency with which I have found them injured, and I may further add that structural changes have been pretty constantly discovered in the convolutional gray substance, when the central ganglia have been damaged. This coincidence has struck me, not only in the cases here alluded to, but also in those of other cerebral diseases. Laborde has particularly called attention to it in his researches on Softening of the Brain.* and with Luys, search for the cause of such concomitant lesions in the arrangement of the radiating fibres connecting the central ganglia with the cortical brain substance.

Much importance has been attached by some authors to the increased weight of the brain in epilepsy, and there is, I think, enough evidence to show that it may be a constant fact. I have ascertained the weight of the encephalon in seven epileptic males, and in eleven

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* Le Ramollissement et la Congestion du Cerveau. Paris, 1866.

females. In every instance there was a difference in the weight between the cerebral hemispheres, but it did occur not always in the same side, for eight times the right hemisphere was heavier than the left, and vice versa ten times. The average weight of the brain proper in males was 49¼ oz.; in females, 45½ oz. Average weight of the cerebellum, in males, 5⅓ oz.; in females, 4½ oz. Average weight of the Pons Varolii and medulla oblongata, in males, 1⅔ oz.; in females, 1⅓ oz. The foregoing results, together with those of Ferrus, Parchappe, Boyd and others, appear to indicate that there is a general increase of weight in the brain of epileptics, if we reckon the average weight of the normal brain to be 43 oz. 13 drs. in males, and 38 oz. 12 drs. in females, as established by J. Reid, upon weighing the encephalon of fifty-three males and thirty-four females, between the ages of twenty-five and fifty-five. This increase of weight acknowledges its cause in the cerebral exudations and hyperplasia of connective elements; yet, unilateral hypertrophy, though a common phenomenon, is not, however, essential to epilepsy, as already manifested by the researches of L. Duchesne.*

The circulatory derangement, impairing regular nutrition, orginates the retrograde cerebral change accidental to epilepsy. Usually, an excessive growth of connective elements attends the atrophy of nervous structure. Not unfrequently, a yellow or orange tint,

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* Gazette Hebdom. de Méd. et Chir. de Paris, 1861, p. 179.

like that of yellow softening, bounds the ecchymotic patches of the convolutions. Such stain, I have seen caused by infiltration of granules of hæmatosine from decomposed blood, as previously noticed by Virchow and Lanceraux. The next is a typical example of these cerebral degenerations.

Case IV. *Epilepsy. Carcinoma of the uterus. Plugging of left middle cerebral artery. Neo-membrane in the arachnoid over left Sylvian fissure. Degeneration of the anterior lobes—superior left marginal convolution and Insula—without aphasia. Pisiform cavities in left centrum ovale, and in right corpus striatum. Lesion of the medulla. Heart enlarged. Neo-membrane in liver and spleen. Pelvic abscess.* (From notes by Dr. F. A. Castle). Plate I.

Julia Q....., aged 37, born in Ireland, by occupation a servant, was a widow, of intemperate habits, and entered the hospital the 9th October, 1866. We could not learn of any hereditary influences, nor the date of the invasion of the spasms, which gradually brought her to the state in which she was when transferred from the Lunatic Asylum, where she had been admitted in 1861. The fits occurred once monthly preceded by vertigo. During them she usually sat down, moaned, did not froth at the mouth, nor bite her tongue, and experienced great trouble of respiration. The convulsions, general, not violent, and attended by incomplete unconsciousness, used to leave her weak and drowsy. She was hypochondriac, complaining a great deal of headache and pain on the left side of the skull. Speech unimpaired, though she kept silent, talking but seldom. Hearing and sight undamaged. Sensibility apparently natural, if there was any change it seemed diminished on the left limbs. Skin dry, temperature normal. Heart enlarged. Lungs with chronic bronchitis. Digestive functions active. She was troubled with metrorrhagia and profuse leucorrhœa. In the summer of 1866, and while at the Lunatic Asylum, she had cholera. She remained very few days at the hospital, often complaining of unremitting headache, and lying all the time on her back, in the most depressed condition. She died in the morning of the 31st of October. She had the last

fit on the 22d of October. Large doses of bromide of potassium, cod liver oil, and expectorants had no decided effect to relieve the obstinate headache, nor the cough and expectoration.

Autopsy on the 31st of October, at one o'clock P. M., weather fine and cool. Body in a moderate degree of emaciation. No rigor mortis. Temperature of the limbs and abdomen 62° Fahrenheit, that of the room being 55°. Pupils dilated, the left more than the right and irregularly. Scalp loosely adhering to the cranium. Calvarium not thick and bloodless. No congestion of the dura-mater. Cerebro-spinal fluid transparent and considerably filling the spaces between the convolutions in the subarachnoid cavity. No congestion either of the pia-mater, excepting around a yellow irregular patch over the left Sylvian fissure, and at the lower part of the lateral surface of the hemisphere, Plate I. Convolutions depressed, particularly at the base of the brain. Pituitary body of a brick color, not enlarged, and remaining in the sella turcica. Cerebro-spinal fluid about thirty ounces in quantity. Weight of the brain, cerebellum and medulla oblongata, forty-seven ounces avoirdupois. Brain proper, thirty and one-half ounces. These organs, the ganglia Gasserii, and the cervical sympathetic, were preserved for microscopical examination.

Melanotic and tuberculous deposits in the lungs. Firm and extensive pleuritic adhesions; enlargement of heart general, though more on right cavities, which, with the pulmonary artery, contained a very large ante-mortem clot. Liver of a dark purple color, not enlarged, adherent to the spleen, with a patch the size of a bean, near the fissure for the gall bladder, moderately distended. Coagulum in ascending vena cava. Spleen indurated, contracted and covered on its upper and posterior surfaces by several small nodules, hard and cartilaginous like, one of them as large as a penny. Peritoneum adhering to the kidneys; the right kidney, of nearer a normal size, upon longitudinal incision, showed a decrease of cortical substance with pelvis and ureter very much distended by urine; left kidney contracted with considerable dilatation of the ureter. Suprarenal capsules reduced almost to a shell. Uterus fixed and firmly united to the bladder, from which it could not be separated without tearing the latter. The cervix uteri and the base of the bladder were the seat of carcinoma, and the surrounding tissue of pelvic cellulitis. There was an abscess in the left ischio-rectal fossa containing about

four ounces of pus. Ovaries not diseased. Mucous membrane of small intestines much softened.

A section of the brain, on a level with the corpus callosum, showed in the anterior part, or knee of the corpus callosum, a small lacuna filled with a glutinous matter of a light grayish color. This cavity extended over the whole width of the transverse commissure. The centrum ovale, in the posterior part of the left side, was stuffed with pisiform cavities, eight in number, of unequal size and bounded by a denser tissue simulating a membrane. They looked like cysts, some as large as a pea, containing a yellow serosity. Two very small similar cysts were located in the right side of the section. In these regions of the centrum ovale, the tissue had the color and appearance of fresh Gruyere cheese. Another lower section, exposing the third and fourth ventricles, and removing the upper part of the optic thalami and corpora striata, manifested in the left corpus striatum an irregular circumscribed stain, of a light brown coloration in the centre, and lilac in the circumference. The convolutional gray substance of the left anterior and middle cerebral lobes was, more than that of the right ones, spotted with small rounded ochre-colored patches, implicating only the cortical layers, with no adhesion to the membrane, over them. This condition was very manifest through the superior marginal, or third frontal, convolutions and the lobe of the Insula, connected by delicate vessels to the superficial patch. The brain tissue seemed bloodless, with puncta vasculosa hardly perceptible. No manifest alteration noticeable in any of the different sections of the cerebellum. Medulla oblongata and ganglia put up in alcohol for microscopical examination. I could ascertain that there was no false membrane in the dura-mater. The patch already noticed in the arachnoid and pia-mater was in relation with the left Sylvian fissure, covering the posterior origin of the superior marginal convolution in an oblong surface shaped like an almond. This patch united intimately to the brain, and on removing it, the gray substance, with a yellowish hue, remained attached to it. The delicate vessels circumscribing the patch, originated from the anterior cerebral artery and the posterior branch of the middle cerebral artery, the anterior branch of which as also a small division near its origin were atheromatous and completely obliterated. The appearance of this adventitous plate resembled

that of those on the spleen and liver, although with these latter, the discoloration was more whitish! They had, however, identical histological arrangement, and their source in the cachectic state of the blood, which Charcot has proved to be not an unfrequent cause of coagula in the blood vessels impairing regular nutrition.*

Passing over the characteristic structure of the carcinoma uteri, I come to the microscopical arrangement discovered in the brain and medulla oblongata. The cerebral tissue was examined fresh, and after hardening in alcohol, to obtain thin sections of its texture. These sections were macerated in alcohol slightly acidulated with acetic acid, and dipped afterwards into pure glycerine until rendered transparent. Let me remark that, such has been one of the plans followed to investigate the minute structure of the nervous system. On other occasions the specimens were previously hardened in a weak solution of chromic acid before putting them in glycerine, or prepared by Lockhart Clark's method. To study the nerve and ganglia, I have macerated them for a day or two in a solution with : two parts of pure acetic acid, one of nitric acid, and three of water. In this way the true nervous elements are hardened, and the fibres of the neurilema and primitive tubes all dissolved. By adopting the plan advised by Sappey,† of boiling the nerve in one part of nitric acid to two of water, we may also isolate the contents of the primitive fibres, but I have found that heat alters the configuration of the elements if they have undergone a fatty degeneration. Liegeois' practice of macerating the specimens in tartaric acid is excellent, as it renders the cellular texture around the nerve quite transparent. Usually, I stained the specimens by dipping them into a neutral solution of carmine; when this is done, the alterations undergone by the capillaries and by the tissue render themselves conspicuous, even to the naked eye, by the darker tint of the stain in the damaged spots. Finally, the preparations have been examined by reflected and by transmitted light respectively, with low and higher magnifying powers of Nachet's microscope.

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*Sur la Trombose qui survient dans certains cas de Cancer, Société Médicale des Hôpitaux, 22 Mars, 1865.

†Mém. de la Société de Biologie, 1862. Tome IV, Troisième Série, p. 88.

The pultaceous substance from the softened portion in the knee of the corpus callosum, was constituted by dissociated nerve tubes and great quantity of granulation corpuscles and fatty moleeules. The capillaries bounding this softening remained free from damage, although masked by the above conglomerated granules. Around the pisiform cavities in the centrum ovale, the fatty degeneration was far advanced. No trace of mycline or nerve tubes, could be distinguished among the connective fibres thickly developed with many nuclei, and their meshes filled by fine fatty granulations and corpora amylacea. The capillary vessels had their walls very granular throughout, this change being mainly in the adventitious sheath of the arteries, described by Robin. (See fig. 1, Pl. VII). I could not make out any membrane lining these lacunæ, nor could I examine any of the serosity they contained. In the regions where the tissue had a yellowish or ochre color, it depended on the infiltration of blood pigment and minute rhombic prisms of hæmatoidine. The appearance of the above cavities, evidently indicated that they proceeded from former apoplectic cysts, and, it only remains to notice that, no pus cells were met in connection with any of them, or the softening in the corpus callosum.

The patches in the corpus striatum and the cortical substance had a similar structure to the one already described in the centrum ovale. But with the latter the nuclei of the connective tissue increased, as also the granules of hæmatosine and the crystals of hæmotoidine, infiltrating the tissue. The capillary vessels, with walls granular throughout, were varicose, easily torn, and stuffed with oil globules and withered blood-corpuscles, in those places in which they intimately united to masses of aggregated dark-colored granules. The patch of the arachnoid over the posterior part of the left superior marginal convolution, intimately united to the cerebral tissue through delicate vessels dipping down into the gray matter. Two branches of the corresponding middle cerebral artery reduced to a firm string were atheromatous and plugged. Such impervious state of the blood vessels, or arterial obstruction, determined the dilatation of the collateral branches, and particularly of those surrounding the patch. This was constituted by the hypergenesis of connective fibres and nuclei mixed to fatty granulations and hæmatic crystals. The gray matter underneath had undergone similar but

not as far advanced degeneration as the previously described. The capillaries had changed here more than in any other place, appearing all granular with partial or general dilatations. These aneurismal swellings reached their maximum in the vicinity of the obliterated arteries; occasionally the adventitious sheath had only dilated upon extravasation through a rent in the degenerated proper walls of the vessel. (Fig. 3, Pl. VII). This state of the cerebral capillaries not unfrequently occurs in chronic epilepsy, producing the punctiform hemorrhage then observed in the gray and white substance. The same vascular change did exist in the cerebellum, very granular and abundant in amyloid corpuscles.

The arteries of the base of the brain were free from atheromatous degeneration, excepting the anterior branch of the left middle cerebral artery, which supplies the pia-mater, and another small branch near its origin, penetrating into the corpus striatum. A fibrinous clot occluded completely the calibre of these arteries; it had an ochre-yellow color, and a fine granular structure, infiltrated with elements of decomposed blood, which, in the pervious portions of the vessel, contained fatty granulations and crystalline plates of cholesterine, mixed with the irregular and granular blood vessels.

The medulla oblongata was harder than the brain. On microscopical examination, different sections exhibited anteriorly no greater lesion than the aneurismal dilation of the capillaries in granular state. The deeper injury showed itself on the vagal nuclei, and transverse commissure forming the posterior boundaries of the cerebral canal, to continue downward into the vesicular columns from which the spinal accessory nerve originates. These regions had undergone an unmistakable fatty change, the capillaries presenting their largest dilatation along the roots of the pneumogastric. Fine brilliant granulations and others with less refractive power, not disappearing with chloroform or ether, and easily removed with nitric acid, or a solution of choloride of calcium, surrounded the ganglionic cells and nerve tubes of the pneumogastric. The lesion had not reached such an advanced stage in the tract of the hypoglossus. Nerve cells in other regions, irregular, dark, and very granular. On the floor of the fourth ventricle, a large quantity of corpora amylacea accompanied the above retrograde elements, and were distinguished again in the inner spur of each vagal nucleus.

It is important to consider these local changes, as the patient did not bite the tongue, and experienced a great distress with respiration during the fits.

The Gasserian and the cervical sympathetic ganglia were not healthy. The former exhibited cells in a very granular condition, with an increased amount of transparent connective elements, still greater and richer in nuclei in the cervical sympathetic ganglia. This hypergenesis of connective tissue, gave a gelatinous aspect to the specimens. Some corpora amylacea had invaded the first ganglion, and in every one the capillaries had participated of the metamorphosis and lengthening described in the nervous centres.

Induration, more or less extensive, of the cerebral tissue has been noticed in epilepsy, and the following is a remarkable instance of induration and enlargement of the cerebral hemispheres.

CASE. V. *Syphilitic Epilepsy. Neuralgic pain, paralysis of the lower limbs. Gummy tumor of dura-mater. Syphilitic deposits with induration, and increased size of the cerebral hemispheres. Complete occlusion of the longitudinal sinus. Amyloid degeneration of the brain and medulla.*

I saw, in consultation with Dr. Perry, of Brooklyn, a ship master, epileptic, who died the 11th March, 1866. Prof. E. R. Peaslee, who had visited the patient in July of 1865, requested me to examine the brain and medulla oblongata. As far as I recollect, the fits occurred within the week preceding Dr. Peaslee's visit, attended with vertigo, temporary palsy, sometimes in one lower extremity, sometimes in both, with neuralgia and most severe pain in the head. The patient suffered from these attacks some years before, but they gradually became less and less frequent, until they ceased altogether. Chancres and inguinal adenitis had also occurred previous to them, and the indications of constitutional syphilis were evident in the skin. When I saw the patient, the fits recurred frequently every two or three days, preceded by neuralgia or violent headache, and followed by paroxysms of wild excitement. I suspected the dura-mater, and most probably the brain, to be involved



abounded, mixed with brilliant fatty molecules and elastic fibres. The capillaries were rare and in a transparent granular condition.

The structure of the cerebral tissue in the circumscribed ulceration varied from that of the rest of the organ. The adventitious growth nearly deprived of vessels, formed the centre of a thick capillary net work in the pia-mater, Plate III. No capillary congestion of the brain; brain substance firm, elastic and pellucid after section. The yellow, cheesy portions of the ulcer had a structure quite similar to that of the dura-mater, but with more fatty elements. The soft, central parts, circumscribed by a denser structure of a dark yellow color, abounded in nuclei, and thick meshes of connective fibres intermingled with fatty molecules, large fatty globules, and a considerable quantity of corpora amylacea. On approaching the apparently uninjured cerebral tissue, the neuroglia increased in nuclei, always mixed with a semi-transparent amorphous matter, and considerable proportion of fatty granulations, corpora amylacea, and scattered fragments of nerve fibres and myeline. The fatty elements were not, however, uniformly distributed and more multiplied in the mass behind the optic chiasma. Different sections of the medulla, manifested the same sclerosis, coincident with dilatation of the capillary vessels, granular and surrounded by heaps of fatty molecules. These varicosities would be discovered likewise in many capillaries around the cerebral ulceration. The increased amyloid corpuscles and nuclei of connective tissue caused the elastic lardaceous appearance of the cerebral tissue. In the medulla the degeneration was strongly marked in the vicinity of the restiform bodies, and in the corpora olivaria. The cells in these latter, had lost their natural fatty aspect, becoming dark and granular.

The lesions in the foregoing case obviously show a circumscribed gummy deposit, coexisting with general sclerosis of the brain tissue, less advanced in the right than in the left hemisphere, both being, however, much increased in size. This enlargement is not common in cerebral syphilis. Virchow reports the extraordinary case of a young prostitute, who

Plate IV.



Ad nat. cop.

Syphilitic Epilepsy.

died shortly after being seized with peculiar nervous symptoms.* *Autopsy.*—Brain with acute hypertrophy. Dura-mater thinned so much as to render visible the cerebral convolutions, which disappeared upon removal of the membranes in consequence of the excessive over-distension of the brain. Small quantity of serosity at the base of the brain and in the ventricles. Cerebral substance firm, but not with the resistance of leather, dry, dense, completely anæmic, and without the least modification in its structure; oblong medulla and nerves originating from it, normal. Dura-mater thickened and reddish at the level of the axis, roots of the spinal nerves uninjured. The autopsy did not sufficiently account for the strange symptoms of the patient. Virchow pronounces the case one of acute hypertrophy, without the least modification in the structure of the brain. This assertion is made, however, without explaining if it is based on microscopical examination, or merely on external appearances as would seem to be deduced from the above record.

Andral† reports four cases of hypertrophy of the brain, irrespective of syphilitic influence: three attended with epilepsy, and the fourth with convulsions in the arms and left leg. Two of the patients manufactured lead paint, and had several attacks of colica saturnina. From the minute description given by Andral, sclerosis

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* La Syphilis Constitutionelle. Paris, 1860, p. 84.

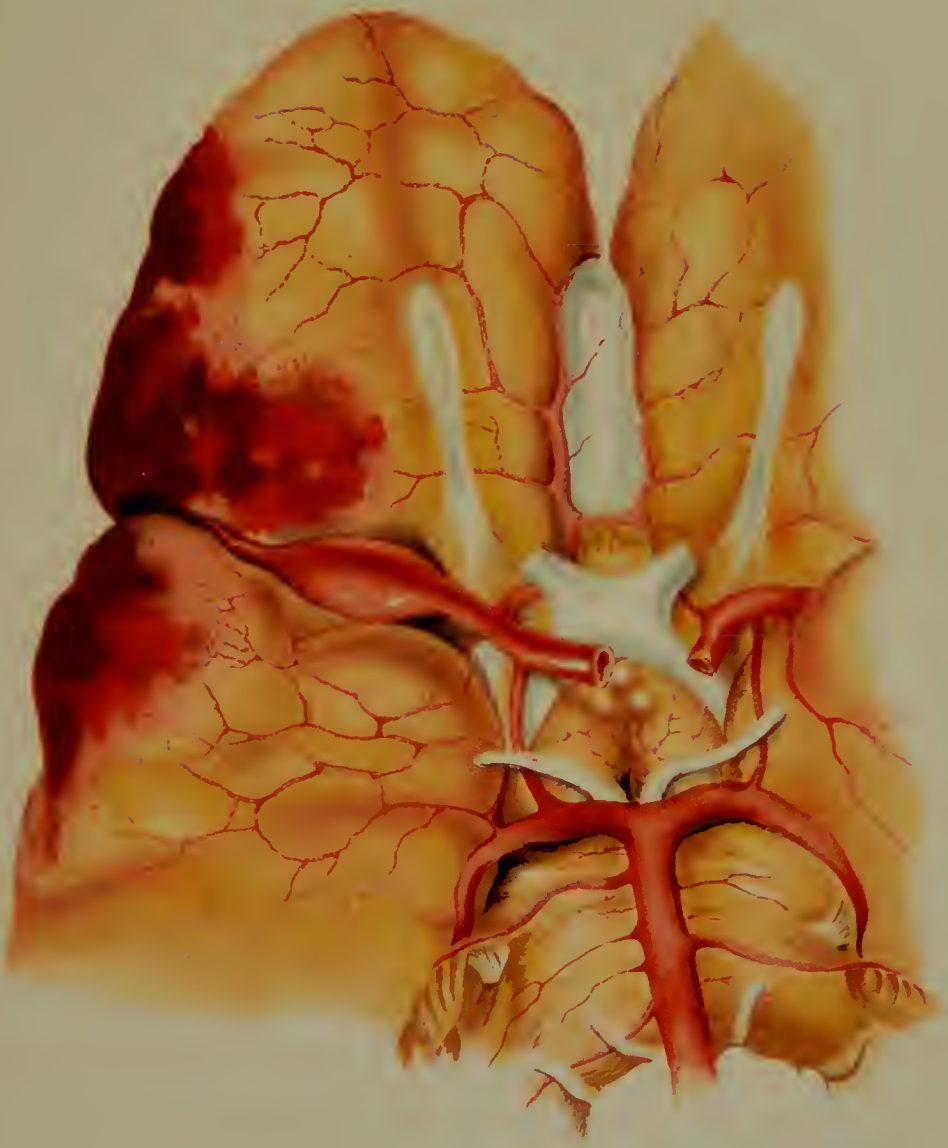
† Clinique Médicale. Tome v, 4ème édit. Paris, 1840, pp. 580, et seq.

must have been the degeneration suffered by the brain, firm, elastic, very resistant and with the meninges very dry. I will describe further on the brain of a congenital epileptic, with general hyperplasia of connective elements in one hemisphere, and atrophy of the other extending to the same side of the medulla above its decussation, and to the opposite below.

CASE VI. *Syphilitic Epilepsy. Ptosis and paralysis of left limbs. Aura from fingers of left hand. Aneurism of the right middle cerebral artery plugged by clot. Sanguineous effusion over the right anterior and middle cerebral lobes. Yellow softening in the right centrum ovale. Sclerosis of right optic thalamus. Miliary granulations in the choroid plexus and ventricular ependyma. Fatty degeneration of the oblong medulla. Tuberculosis of lungs. Heart normal. Plate* IV.

Louise F.... Irish servant girl, aged 26, came under my care through the dispensary of the St. Catharine's House of Mercy. She had constitutional syphilis; engorgement of the cervical ganglia, roseola, patches in the genitals, and leucorrhœa, obstinate headache and epilepsy with ptosis and paralysis of the left limbs. The antisyphilitic treatment with the iodide of potassium, proto-iodide of mercury, and cod liver oil, proved of no more avail than to remove the cutaneous accidents. The epileptic attacks continuing with troublesome coughing, vomiting, and other nervous symptoms, the intellectual faculties gradually failed, and, running into a soporous condition, the patient finally died in a fit, the 12th March, 1863. In the commencement, an aura starting from the fingers on the left hand, preceded the paroxysms, but subsequently this warning ceased. During the fits the girl used to bite the tongue, and to froth considerably at the mouth.

Autopsy.—Calvarium normal, vessels of the diploë very congested, meninges opaque. Serous effusion in the arachnoid cavity: this membrane opalescent on the anterior part of the base of the brain. Right middle cerebral artery plugged by a firm, laminated, orange-tinted clot, adhering to the walls and causing aneurismal dilatation



Ad nat. cop.t

Aneurism of the left middle cerebral artery.

of the vessel. A large surface of the anterior and middle lobes in the vicinity covered by a film of coagulated blood, and the gray matter underneath, infiltrated and softened, without delimited boundaries with the white substance somewhat yellowish in this region. On section the brain tissue looks rather wet, with two yellow patches the size of a hazel nut, in the right centrum ovale, near the corpus striatum, and surrounded by softened tissue. Ventricles containing turbid serosity; choroid plexus rough, covered by small, firm, yellow granulations. These miliary bodies covered also the ependyma of the fourth ventricle, and the pia-mater over the medulla. Both lungs with tuberculous deposits at the apex, extensive pleuritic adhesions on right side, with gray hepatization of the lower lobe. Heart natural, without any valvular lesion. The other viscera presented nothing worthy of notice, excepting the liver transversed by fatty streaks. Uterus enlarged — granular cervicitis. Mucous patches on labia minora. Left median and ulnar nerves removed for examination. The granulations in the lining membrane of the ventricles contained a cheesy matter, easily pressed out from an envelope formed by delicate connective fibres. Large oil cells and granular nuclei mixed with fatty particles, composed the above matter. Same fatty metamorphosis repeated in the patches of the centrum ovale, with more or less addition of neuroglia and nerve fibres or cells in granular disintegration. The softened tissue around contained nervous elements fragmented, large granular corpuscles, and amorphous matter. The gray matter under the apopletic effusion showed no nervous elements among the granular cells, fatty granules, and hæmatic crystals and globules constituting its softened structure. While such softening and transient stages of retrograde metamorphosis were thus evinced in these regions of the brain, the left optic thalamus and the cervical sympathetic exhibited a typical sclerosis, with its characteristic exuberance of connective elements. In the oblong medulla, the fatty degeneration existed principally, on the left side, along the path and nucleus of the hypoglossus, and hence extending to the floor of the fourth ventricle. The vessels, distended and granular, were masked by fine, fatty granulations, grouped along their course. They underwent the same but less general alteration in the brain and cerebellum. The latter exhibited

an increased amount of connective nuclei and amorphous matter stuffed with fatty molecules.

The change in the roots of the hypoglossus, was remarkable. Primitive fibres reduced to their sheath and cylinder axis, medullary substance lost or fragmented into fine brilliant granules. This lesion had also invaded many of the primitive fibres in the roots of the right pneumogastric — the majority of the abducens and those of the sympathetic. No change in the median and ulnar nerves.

This case is a fair example of the retrograde metamorphosis that may occur in syphilitic deposits of the brain. There can be no doubt that the plugging of the middle cerebral artery determined the apoplectic effusion with disorganization of the convolutional gray matter. Such morbid accident occurred without evidence of cardiac disease to account for the arterial coagulum, with a laminated structure, and giving rise to the aneurysm of the middle cerebral artery. Dr. Bristowe has reported* a case of obstruction by clot of the right middle cerebral artery unattended with heart disease, in a patient who had been suffering from secondary syphilis and in whom the cerebral tissue was softened in the corresponding part of the middle lobe. The same author had before shown the occurrence of this important trouble of the circulatory system in connection with secondary syphilis.† A woman with secondary syphilis was at different times seized with four epileptiform attacks — the third attended

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* Medical Times and Gazette, Nov. 19. 1864.

† Transactions of the Pathological Society of London, vol. x, p. 44.

with hemiplegia, and the last with coma and death. Heart normal. The two internal carotids obstructed by clot, and both anterior cerebral lobes and the corpus striatum in the right side softened. Reference has been previously made to the influence of the cachexiæ in determining the formation of coagula obliterating the blood vessels, and the same cause, without necessity of cardiac trouble, may have acted in the above cases.

This instance affords beside evidence of the connection between pulmonary disease and injury of the medulla oblongata; a coincidence pointed out by Van der Kolk* in three out of seven epileptics who had bitten the tongue and who died with phthisis pulmonalis, and also in a fourth case where there was emphysema of the lungs. One of two other uncertain cases died likewise with phthisis. Among seven epileptics who did not bite the tongue in the attack, one died with pneumonia; with the remaining, the condition of the lungs was unnoticed. My results and those of Van der Kolk agree in what relates to the connection between pulmonary disease and injury of the medulla. But, in addition, I have met with pulmonary lesion when the path and origin of the pneumogastric were damaged, whereas Van der Kolk particularly refers to such a coincidence in those patients who did bite the tongue during the fits, and showed greater damage along the hypoglossus. In the table of patients, who did not bite the tongue, and

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*Op. cit., p. 245.

in whom the degeneration predominated in the region of the vagus, the state of the lungs is mentioned but once. In close relation to this subject I may say, that among my patients who died in or immediately upon an attack* all but two were those in whom the degeneration displayed itself more markedly in the vicinity and origin of the vagus; a result confirmatory of that of Van der Kolk and Kroon, who assert: that those patients who did not bite the tongue had died in a fit. Brown Séquard,† quoting cases from Jobert de Lamballe, Stuart Cooper, and Rostan, as also Van der Kolk,‡ have reported examples of disease of the Pons Varolii and the medulla oblongata involving the vagus, and attended with pulmonary trouble. I need not recall the very interesting experiments of Schiff to demonstrate this influence of the pneumogastric. With the case already related, as well as in others in which the origin of the pneumogastric was injured, and the patients had not bitten the tongue during the fits, I have usually met with some thoracic lesion — generally tubercles — accompanying the epileptic affection. I could mention likewise instances in which I have watched the development of the pulmonary disease, as the epilepsy progressed, the patient of course not biting the tongue during the at-

* See Synoptic table at the end of the chapter.

† Journal de la Physiologie de l'Homme. Tome I. Paris, 1858, pp. 526, 532, 760, and Tome II, 1859, p. 723.

‡ Case of Atrophy of the Left Cerebral Hemisphere. New Sydenham Society, London, 1861, p. 171.

tacks and thus giving ground for conjecturing that the pneumogastic was involved. I have recently examined the pneumogastric and cervical sympathetic from a child who died with hooping cough, and compared the changes they exhibited with those of the same organs from another epileptic child, both patients at Randal's Island Hospital, directed by my friend Dr. F. A. Castle, who kindly sent the specimens to me. In the first case the connective fibres and nuclei and the capillaries, were very much increased in the ganglia and nerve, their condition being that of neuritis; in the latter instance, however, the hypergenesis of connective elements was not so high, and the cells and primitive fibres had undergone beside a distinct fatty degeneration. In investigating the pathological changes of the sympathetic system and nerves connected with it, I have met with redness and enlargement of the pneumogastric in cases of pneumonia: The medullary substance has appeared broken up in minute fragments, and the external membrane of the primitive fibres, as well as the perineurium, displaying a great abundance of nuclei. This change I noticed most particularly in one pneumogastric brought to me by my friend Dr. J. B. Done, and removed from a patient who died with pneumonia at Bellevue Hospital. In phthisis — and chiefly with children — there is a peculiar degeneration of the pneumogastric and cervical sympathetic ganglia. Independently of the fine fatty granulations contained in the tubes and nervous cells, or between and underneath the fibres of the perineurium,

we find in such instances a multiplication of transparent fibres of connective tissue with large oval nuclei, in many places pressing the medullary substance out of the tube and giving to the nerve fibre a varicose appearance. This hypergenesis of connective tissue, with identical characters, occurs in the ganglia, and it is sometimes so great as to increase the size of the nerve, making it look hypertrophied. The red semi-transparent discoloration of the tissue is due to the presence of the above described nuclei.

The fatty degeneration of the brain becomes still more striking in cases of epilepsy originated by chronic alcoholismus. I have described some time ago the fatty change of the brain in delirium tremens.[1] In simple cases the lesion does not go beyond a determination of blood, which may, however, induce an acute inflammation of the brain. After repeated attacks, the sanguineous determination gives rise to stasis of blood, with dilatation of the vessels and exudations, impairing the structure of the organ, as also originating a decided subacute inflammatory condition, which brings at last acute peri-encephalitis, epilepsy, progressive paralysis, or dementia. The degeneration of the blood-vessels is very prominent on these circumstances; not only they are lengthened and distended, but frequently plugged by clots. Their adventitious sheath becomes granular, and fine fatty molecules are

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[1] *On the Proximate Cause of Delirium Tremens.* American Medical Times, vol. IV, No. 19, May 10, 1862.

piled between it and the walls proper of the vessel which, on the later stage, participate also of the degeneration. The vascularity of the cerebral tissue, no longer uniform, renders it anæmic in those regions where the metamorphosis has reached its maximum. On slicing the cortical substance, it may exhibit in many points a brown or violet discoloration, often traversed by yellow streaks. It is unnecessary to say that these patches are remnants of apoplectic effusions, and that the fatty elements are here mixed with hæmatosine and hæmatoidine.

The most extensive fatty metamorphosis of the brain and blood-vessels I met with was in this next instance.

Case VII. *Epilepsy from intemperance. Right hemiplegia. Aphasia. Ventricular hemorrhage. Fatty degeneration of vessels in the choroid plexus, and of the middle cerebral arteries. Fatty degeneration of the encephalon, greater in the anterior cerebral lobes.*

An intemperate epileptic old woman died at the Alms House, Charity Hospital, in September, 1866. I saw her three or four days before her death. She had been seized with violent attacks the previous night, upon which she became hemiplegic, or I rather say, had contraction of the right limbs, and aphasia. She could understand what was asked, but unable to articulate any answer. Any attempt to protrude the tongue, or to move the limbs, increased their muscular contraction. The cheeks were flushed, the left pupil contracted, tongue dry and furred, pulse slow, radial and temporal arteries hard and unyielding. I diagnosticated hemorrhage in the cerebral ventricles, probably from rupture of a fatty vessel, and made the most unfavorable prognosis, expecting that a renewed attack would determine an exacerbation of the symptoms with greater extravasation, terminating her existence. This soon occurred, for that very night she died in a violent fit. I was not present at the

autopsy made by the resident physician, Dr. D. W. Searle. He was, however, struck with the pale condition of the brain on removal of the dura-mater, as he anticipated finding a general congestion of the organ. I have no exact recollection of the lesions in the thoracic and abdominal viscera. Two days after the autopsy, on my visit to the hospital, I examined the brain and medulla, carefully preserved in alcohol by Dr. Searle. The bloodless state of the pia-mater, and of the cerebral surface was very notable. On section, the brain tissue dry and yellowish, disclosed irregular patches of punctiform injection. The ventricles were largely distended, and filled with clotted blood. The clot in the left side, fibrinous, firm and shaped to the lateral ventricle, evinced its older formation. The septum lucidum had been destroyed, and the cavity of the right lateral ventricle filled with grumous blood, recently extravasated, and which had reached the third ventricle through a large rent of the velum interpositum. The blood vessels of the choroid plexus were fatty and friable, the cerebral arteries on both sides had undergone a general fatty degeneration. The left middle cerebral artery in places contracted, but in others distended, had lost, in other points, all resistance, and could be easily torn. Under the microscope the brain tissue and that of the cerebellum displayed a general fatty degeneration, deeply involving the gray matter of the anterior lobes, the whole left locus niger, and a large portion of the right in the crura cerebri. Different sections of the medulla oblongata, manifested an advanced alteration in their minute structure. The ganglionic cells, darkened and granular in the origin of both pneumogastrics, did not appear so much so in the nuclei of the hypoglossi. It could be distinguished very plainly that the greatest dilatation of the capillaries existed in the vicinity of the vagal nuclei. In conclusion I may add, from the memorandum with the history of the specimen, that no cicatrix was noticed in the tongue. It was also learnt from the companions of this woman, that she at night, after the fits, remained delirious and raving for some time.

Delirium, the not unfrequent sequel of epilepsy, is more habitually met with when the disease is caused by alcoholismus. It has been a common symptom

with the large number of epileptics admitted into the hospital, and with whom intemperance has counted as an efficient adjuvant, when not as the primitive cause of the disease. I, of course, am not referring to any case of epileptic convulsions happening along with delirium tremens, but solely to epilepsy originated by intemperance and outbreaking without the last concomitant trouble.

Let me pause awhile to make some remarks on the cause of delirium, and especially applicable to that in *mania à potu*. Whatever its origin delirium is the result of cerebral hyperæmia. This statement is at variance with the generally received opinion which considers some of the cases as the result of anæmia of the brain. Authors admit that, when delirium supervenes upon a debauch, it is due to cerebral excitation or hyperæmia, and to cerebral exhaustion or anæmia when it occurs upon the withdrawal of the alcoholic stimulus, in those persons accustomed to indulge freely in drinking. This latter statement is, indeed, utterly opposed to what we learn from a close investigation of the brain; for cerebral hyperæmia is the proximate cause of delirium in the first as well as in the second case, in which it happens in this wise. The increased activity of circulation from constant alcoholic stimulus, gives rise to a lengthening and dilatation of the cerebral blood vessels; now, as soon as the withdrawing of the stimulus diminishes the force of circulation, a stasis of blood takes place, and hence cerebral hyperæmia, the true source of the mental disturbance. More-

over, we may see that by reference to the symptoms, we are enabled to suspect this very nature of the modification undergone by the brain. The attentive examination of an intemperate man, at once shows that beside his peculiar stammering, there is a manifest tremor; he may be able to control his movements, but never his constant shaking. These phenomena, even slight, afford undoubted evidence of the congestive state of the brain, and, I may assert that, they are always premonitory of the delirium. Tremor is a symptom most constantly associated with a sub-inflammatory or hyperæmic chronic condition of the nervous centres; therefore, it usually accompanies softening of the brain, paralysis from hemorrhage, chronic meningitis, induration of the nervous centres — the result of exudations produced among the elements of the organ, and all slow intoxications attended with cerebral congestion, such as those from mercury, opium, cannabis indica, etc., etc. Having thus already accounted for the hyperæmia in delirium following withdrawal of the alcoholic stimulus, and keeping constantly in sight the morbid changes of the vessels hindering regular nutrition, it is easy to see how such a relation of cause to effect should always exist. But, it may be argued that exhausting conditions are apt to bring on delirium, and on this category comes epilepsy. It must be, nevertheless, remembered that it is a law of pathology, that deficient nutrition is the ordinary source of sudden local congestions, and hence we find that delirium is often a relapsing symptom in

the convalescence of protracted fevers, or after epileptic attacks recurring at short intervals and thereby impairing the nutrition of the brain. Nothing is more common than cerebral congestion with anæmia, chlorosis, or with rheumatic, syphilitic, cancerous or any other cachexiæ. Yet, it may be argued again, that under the latter circumstance delirium is the effect of irritation by some adventitious growth in the brain. It must, however, be borne in mind that an adventitious growth interfering with the brain does not produce delirium unless congestion or inflammation be more or less extensively developed in the brain tissue connected with the adventitious one. Even in cerebral atrophy, commonly attended with chronic delirium or mania, the hyperæmic state of the brain is evident in the lengthening and dilatation of its blood vessels.

I have, as previously advanced, studied the nervous centres in two cases of epilepsy attended with idiocy. The morbid changes in these occasions, as with epilepsy generally, have been of an atrophic nature — diminution of cortical substance and of the nervous elements, with exuberant genesis of connective tissue undergoing a retrograde or fatty metamorphosis, such may be resumed the kind of degeneration displayed by these cases. One of them, however, is too interesting not to occupy a place here, and will be the last I shall report in reference to the lesions manifested by the brain in epilepsy.

CASE VIII. *Congenital epilepsy. Idiocy. Paralysis and contraction of right limbs. Atrophy of left cerebral hemisphere, left anterior pyramid and of right half of the spinal cord. Tuberculosis of the lungs. Stricture of the colon.*

A young man, aged about twenty, epileptic from infancy, incapable of articulating but a few meaningless words, and wandering all the time around the grounds of the Alms House, was in my division at Charity Hospital. He showed a peaceful disposition, very easily manageable, and had paralysis with rigidity of the arm and hand, and talipes equinus, with shortening of the upper and lower right limbs. The epileptic attacks had decreased in frequency when towards the early part of June, 1866, he took to his bed and gradually sank in a low condition, without fever or any marked evidence of visceral derangement. The autopsy was made by Drs. L. M. Yale and F. A. Castle, resident physicians. Body emaciated, but with flesh of the right arm and leg remaining more than in the opposite limbs. Skin of the back and extremities with scattered pustules of ecthyma. Excepting pulmonary tuberculosis and stricture of the colon, nothing else particularly observable in the thoracic and abdominal viscera. Calvarium regularly shaped, thick, pale, scanty in diploë, with a quite smooth inner surface, measuring ten and three-quarter inches in an antero-posterior, and eleven inches in a transverse direction, and twenty inches in circumference. Dura-mater bloodless, not adhering to the bones. Vessels of the meninges emptied; cavity of the arachnoid filled by pale serosity. Brain and cerebellum weighed thirty-nine and a half ounces, and the first alone thirty-three ounces. The convolutions flattened on the left hemisphere, projecting some on the right, the cerebral surface appearing tinted with a transparent gray yellowish hue. The left hemisphere was of smaller size than the right one. On section through the corpus callosum a difference was found of half an inch between their respective lengths. Ventricles distended with serosity, choroid plexus colorless, ependyma and pineal gland softened, cavity of the right ventricle larger than that of the left. The same inequality noted with regard to the right optic thalamus and corpus striatum, not existing, however, with the tubercula quadrigemina and the cerebellum, which kept their symmetry unimpaired. The unilateral

predominance on the right manifested itself again in the Pons Varolii, and the anterior pyramid, more prominent and larger than the congener, the olivary bodies, on the contrary, being of equal size. No want of symmetry in the spinal cord, but the right ganglionic roots were slighter than the left in all the nerves down to the middle of the dorsal region. No difference in the size of the Gasserian ganglia. A rugous, hard, cretaceous patch occupied the posterior part of the left centrum ovale, near the superficial cortical substance, covering as large a surface as that of a silver three cent piece, and the same but punctiform degeneration was discovered along the extraventricular portion of the corpus striatum, and the tænia semicircularis.

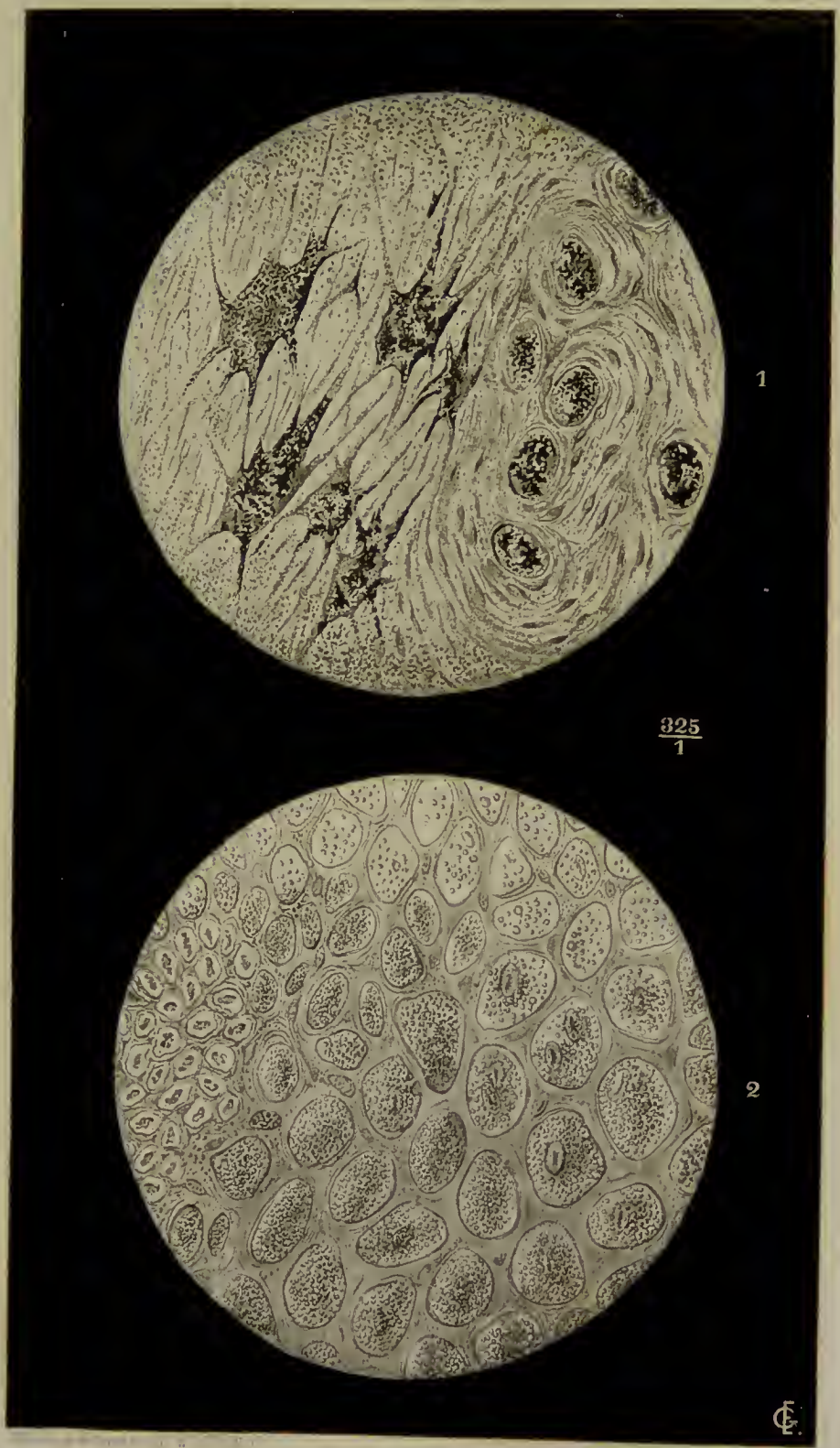
In the left side of the base of the brain the convolutions were effaced, the bulb of the olfactory nerve deficient, the perforated space gelatinous, optic tract smaller; thereby rendering the posterior part of the commissure unsymmetrical, but without disparity of the optic nerves, and the crus cerebri thinner than the right. Although I made no special measurement, a lack of growth manifested itself conspicuously in the bones of the limbs and of the corresponding half of the pelvis, this being broader and more bulky in the left side.

Portions of the muscles of the anterior tibio-fibular, and of the radial and posterior brachial regions, with the respective nerves of the deformed limbs, were saved for microscopical examination. The spinal cord with all its roots, the cervical sympathetic, and the semilunar ganglia of the solar plexus also were preserved.

The minute structure varied in the cerebral hemispheres. The left had changed into a mass of fatty tissue in its highest degree of retrograde degeneration. The cretaceous patch and points in the centrum ovale, contained degenerated capillaries with walls encrusted with fine calcareous granulations, also collected in clusters of globular masses between the fatty elements, and entangled in an imperfect net work of connective tissue. This arrangement seemed to mark the sites of primitive congestions with increased vascularity of the parts, probably engendered by some local inflammatory process in these regions of the brain, which possibly took an active part in the etiology of the cerebral agenesia. The metamorphosis of the right hemisphere was of quite another kind and displayed a rank growth of connective fibres and nuclei with atrophy of the nervous elements.

This, indeed, must have been the prior stage through which the congener hemisphere passed at some earlier age, for such hyperplasia of connective elements is the forerunner of atrophy or fatty change, as evinced by its commencement in the central ganglia of this very same hemisphere. The exuberance of the connective tissue and nuclei gave the gray pellucid appearance to the brain tissue. The cerebellum participated as much of the degeneration, but with a greater preponderance of nuclei. As to the capillaries, deficient in number, they had undergone all over the encephalon a fatty or the above described calcareous degeneration.

The medulla oblongata had suffered extensive change. The withered nerve fibres, fragmented or granular, were compressed by an excessive amount of connective tissue and fatty granules. Few had escaped injury in the right pyramid, and none existed in the left, but were transformed into a heap of connective fibres intermingled with nuclei and fat. This absence of nerve fibres continued upwards through the corresponding half of the Pons Varolii and the crus cerebri, and downwards it mainly involved the left inner boundaries of the anterior spinal fissure, to display itself again more conspicuously in the posterior region of the right lateral column. Nervous elements, more spared in the midst of similar atrophic degeneration, and stained with pigment infiltration, in the corpora olivaria. Some capillaries of the medulla oblongata exhibited a calcareous change, but the greatest number were fatty. The floor of the fourth ventricle, the tissue of the pineal gland, and of the ependyma in the cerebral ventricles, appeared thickly interspersed with brilliant corpora amylacea, also met with in the spinal cord and medulla. The cord had not been equally involved throughout by the lesion. A conspicuous absence of nerve fibres noticed along the cervical and upper portion of the dorsal regions in the right anterior and lateral columns, strongly contrasted with their larger proportion in the corresponding regions on the left side. This difference in the amount of fibres coincided with that manifested in the calibre of the right spinal nerve roots. In the above named regions, a transparent gelatinous degeneration, with infiltrations of brown pigment granules, surrounded the central canal, encroaching in some places for a great distance on the gray substance. This, very much disfigured on the right side, preserved a more natural


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shape on the left. In the cervical and lumbar enlargements the anterior columns showed, in the right side, an areolar fibrous structure filled with oil globules, they were almost disorganized and gelatinous between the cornua, and completely destitute of nerve fibres. The connective tissue had less increased in the posterior columns, wherein shrivelled nerve fibres could be made out: in this condition, they would be distinctly traced from the dorsal region upwards into the corpora restiformia, to be there confounded with the atrophied fibres of the lateral columns. The majority of nerve cells, although disfigured, remained in other respects unimpaired on the left half of the cord, but the tissue through the whole organ contained great amount of brilliant amyloid corpuscles, and granulation cells filling the above described areolar spaces, or, in other places, as it were, in an early stage of formation, exhibited by the irregular collection of molecules more or less approaching the globular form, fig. 2, Pl. v. Throughout the dorsal region, the gray and white substances looked not so deeply hurt, and a general granulo amyloid degeneration had invaded the whole conus medullaris.

The sympathetic showed its ganglionic cells broken up or shrunk, or those spared, completely infiltrated with brown granules concealing their nuclei. They had been atrophied by the unnatural increase of slender, nucleated transparent fibres, fatty granules, and amyloid corpuscles, found beside in the spinal lumbar ganglia. Many of the attenuate nerve fibres, reduced to their sheaths, had lost their contents, or partially retained them in a granular state, but only rare ones exhibited a healthy form. Many oval nuclei, like those of the connective fibres, could be distinguished in the tubes destitute of cylinder axis. The semilunar ganglia, grayish and gelatinous, were composed of exceedingly delicate fibres, having distinct nuclei scattered in them. Very few complete cells could be made up in the middle of a considerable quantity of pigment and fatty granules entangled in the meshes of the above fibres.

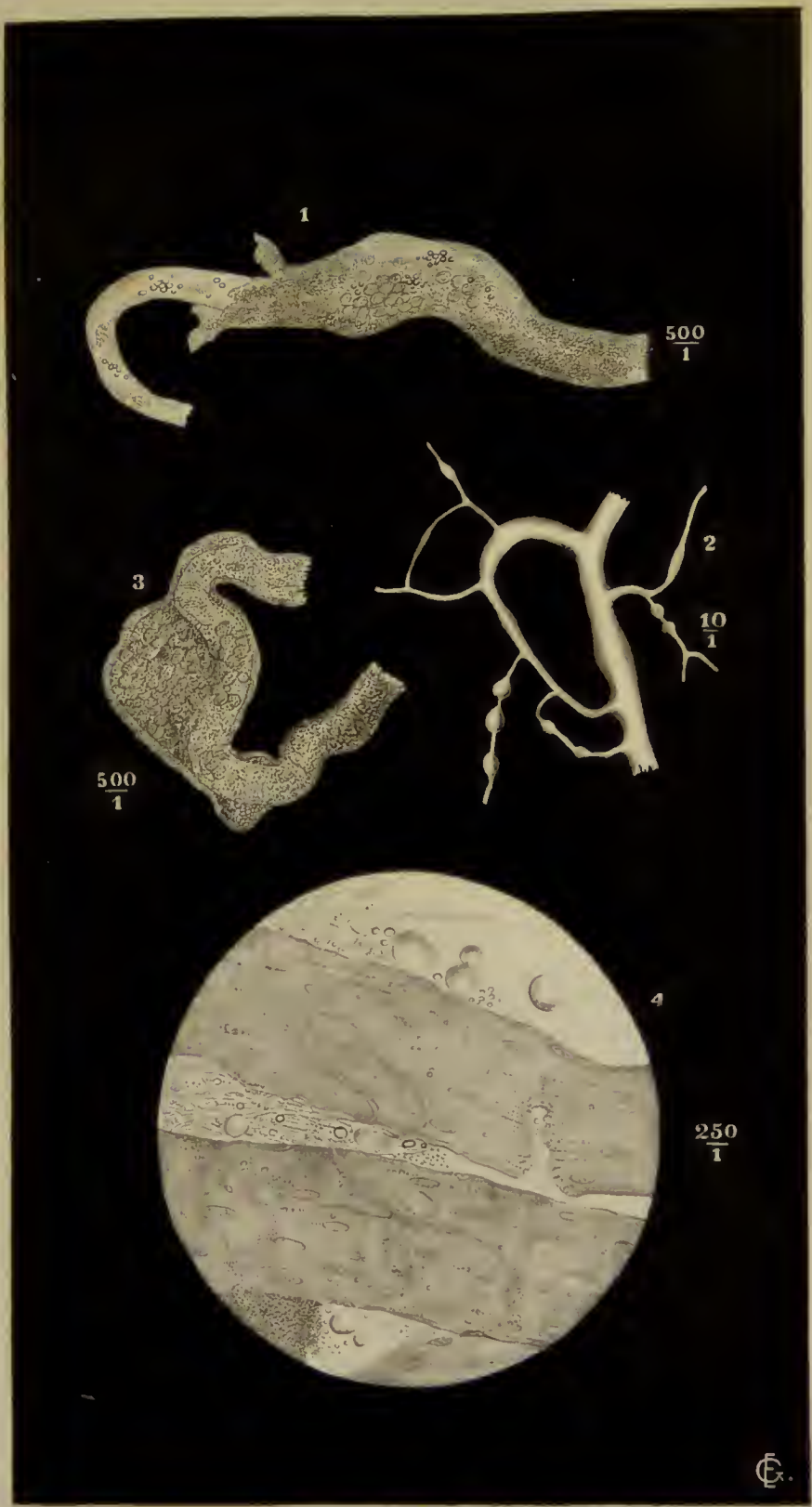
I studied most carefully the muscles of the palsied extremities. In many I discovered a fatty degeneration, replaced in others, by a great abundance of connective tissue, the primitive muscular fibrils being thus rendered gelatinous, transparent, and with several nuclei of elongated shape in the sarcolema. (Fig. 4, Pl. vi). Occasionally both alterations coexisted in the same muscle. The size

of the muscles, larger than those on the uuinjured limbs, only resulted from such a hypertrophy of connective tissue, or muscular sclerosis.* The nerves of these limbs, rigid during life, in consequence of paralysis, lost a majority of their primitive fibres, the remaining considerably diminished in calibre, filiform, with irregular outlines, partially contained in some points of their length fragments of myeline and cylinder axis, in granular disintegration. Finally, the blood vessels of these parts did not keep free from injury, for they had undergone, in the capillaries connected with the pustules of ecthyma, a distinct fatty metamorphosis.

I could not discuss as they merit the important points related to this case. The most interesting is the manner the injury extended from the brain into the spinal cord; originated in the left hemisphere, it continued through the same side of the Pons Varolii and left pyramid to reach, below the spinal commissure, the posterior region of the right lateral column. It is true that no inequality could be perceived in the spinal cord, but on microscopical examination, the right and not the left half, particularly in the antero-lateral columns, had principally suffered the influence of the atrophic metamorphosis of the opposite upper cerebral regions, which did not seem so far advanced in the posterior columns, nor in the olivary bodies. Van der Kolk in his remarkable monograph on

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*This degeneration corresponds with that described by Duchenne, de Boulogne, in *Pseudo-hypertrophic-paralysis*. Arch. Gén. de Méd., January to May, 1868. See also my paper *On Treatment of Paralysis by Hypodermic Injections of Strychnia, with Remarks on Infantile Palsy*. Proceedings of the Connecticut Medical Society, 1868, p. 114.


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Atrophy of the Left Cerebral Hemisphere,* is inclined to believe, on the description of cases published by other authors, that the corpora olivaria are less directly related to the cerebrum than the corpora pyramidalia, and this view is likewise sustained by this case. As to the cerebellum, though degenerated in structure, it did not present any unilateral deviation from regularity. To the hyperplasia of connective elements, was due the unimpaired symmetry of the cord. If further evidence of it were needed, it may be afforded by the slighter size of the spinal nerves in the right side. Such atrophy of primitive fibres beneath the decussation of the pyramids on the side of the cord opposite to that of the atrophied cerebral hemisphere, is particularly noticed by Van der Kolk, who appears to be the first to observe it, although he remarks that according to the striking difference represented in the drawing of the specimen it must have existed in a case reported by Weber.

It is equally curious that the antero-lateral columns were destitute of nerve fibres, by no means so totally destroyed in the posterior columns, the degeneration of which was distinctly traced from the dorsal region into the corpora restiformia, thus confirming the anatomical relationship of these regions of the nervous centres. This complete atrophic change of the left anterior columns contrasts with the less injured state of the posterior columns, and seems to indicate that

* New Sydenham Society, London, 1861, p. 149.

the brain does not exert any prominent influence on the nutrition of the sensitive fibres, the trophic power of the ganglia being on the contrary sufficient to maintain it along distant ways upwards in the posterior columns. Conversely, the downward influence of the brain on the motory fibres of the anterior roots accounts for their preservation on the left side and disorganization or deficiency on the opposite, naturally reducing the magnitude of the spinal ganglia. Without disregarding the trophic power of the latter, I believe that the morbid changes discovered in the sympathetic must have originally acted, causing the impediment to nutrition, attended with the general metamorphosis evinced by the nervous and other anatomical elements. Therefore, to this original lesion, of the sympathetic should be attributed the paralysis and diminished development of the bones in the right limbs. Van der Kolk asserts that in his case, and in ten out of twenty-seven others of unilateral cerebral atrophy, the half of the skull corresponding to the affected hemisphere was increased in thickness. Such difference manifested itself in no positive degree in the case under consideration, although the bones of the skull were rather hypertrophied.

As usually happens, the rigidity and incomplete paralysis were greater in the arm than in the leg. From the instances on record, it is not proved, as Van der Kolk has already said, that the most paralyzed are the most atrophied limbs in unilateral atrophy of the brain. With my patient, the arm

retained larger dimensions than the leg, both having a fleshier appearance than the opposite; but such a greater bulk of these limbs, as previously manifested, depended on the myosclerosis or pseudo-hypertrophic paralysis of Duchenne, displayed by the muscles. Paralysis and contraction commonly accompany each other in unilateral cerebral agenesis, as shown by the cases of Van der Kolk, by three others reported by Dr. J. W. Ogle,* and by the present. The unsound condition of the cerebral gray matter accounts for the lack of intellectual faculties, which prevented any accurate investigation concerning sensibility in this case. Nevertheless, tactile sensibility was not lost, for the patient, when touched, willingly withdrew the limbs, which finally exhibited an eruption like scattered ecthyma, also existing on the face and back.

Reference has been made in the preceding pages to lesions disclosed by the cerebellum in the cases already reported. I have not so far observed any constant localization indicative of a connection between the parts injured and those affected in the medulla oblongata. Twice the degeneration has appeared mostly in the corpus dentatum; with the remaining instances it has irregularly shown itself in the white and gray substances of the cerebellum. As to its nature, it has in every instance been the same as in the brain, with greater increase, however, of connective nuclei, or *myelocites* of Robin. I have, in addition, more frequently

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* Med. Times and Gazette, 26 Nov., 1864.

found the cerebellum injured than the brain. Such have been the general results, and, as they may be wanting in distinctness, it will be worth while to add the report of a very remarkable instance in which one cerebellar hemisphere proved to be involved by a rather uncommon lesion.

CASE IX. *Epilepsy since infancy. Tottering gait, hyperæsthesia of the skin. Cerebral congestion. Cholesteatoma in left cerebellar hemisphere. Lesion of the medulla, and of the gray spinal substance.*

A girl, aged 12, born in Waupaca, Wisconsin, entered the Mahopac Medical Institution for epileptics and paralytics, April 7, 1868. She was slender, pale, slightly developed for her age. A brother of her maternal grandmother became insane upon injury to the head, and her father died with unknown disease, causing extreme emaciation. She had the first convulsions when six months old, while nursing and teething; the spasms were the sequel of dysentery, which, attended with fever, had previously affected the mother. Convulsions were a symptom thereafter present each time that the child was sick from any cause whatever, became more and more frequent, and, three years ago, assumed an epileptic character without any appreciable cause, unless it be the change of climate from Wisconsin to Pennsylvania, where her family resides. Has had measles and other diseases of childhood, each one attended with paroxysms of convulsions when it reached its height. When about five years old she had scrofulous enlargement without suppuration of the lymphatic glands of the neck. In the beginning, the attacks were limited to loss of consciousness, occurring two or three times a day, then ceasing for several weeks, and happening generally at night, but latterly not as much so as in the early part of the winter, during which season she appeared always worse. At first the convulsions seized her suddenly, now she feels sick at their approach "with something coming up her throat," frequently asks "What shall I do?" and almost always screams after turning very pale, and falls immediately backwards. The heart beats also violently, and the pupils are

largely dilated before the attack, during which she froths very little at the mouth, bites the tongue and lips, and is much convulsed. A year ago, urine was passed for a few times during the attack, which does not now take place; nor does she wet her bed at night. Occasionally, she is irritable before the fits, and when they repeat she becomes very wild, and bites and scratches herself or the persons near her. She cannot stand, not walk after the attacks, and staggers on taking her first steps. Between the fits she feels as though she were going to be taken sick, becomes nervous, with palpitation of the heart, and suddenly runs away from the house without paying attention to any one calling her. Once she went a mile before being caught, and on another occasion she escaped through the window of the room where she was. Her speech is thick after the fit. There has never been any dysphagia, nor impairment of sight, nor unequal dilatation of the pupils. When not drowsy, after the attacks, she talks wildly and sleeps seldom over half an hour, but always does so between successive paroxysms. In the intervening period between the fits, she bites and scratches herself as already noticed, without consciousness of what she is doing; but otherwise she is so sensitive that the least motion or touching of the limbs causes her great pain. The temperature is lower on the right than on the left side of the body: she perspires freely and chiefly on the right side. Formerly she had a voracious appetite after the attacks, but now she goes without eating anything.

The breast has been enlarging for more than a year, and there has been more or less tenderness in the loins and back. Her sisters menstruated at about 12 years of age, but she has not yet.

When she arrived at the Institution she was in a very excitable condition, having had eight attacks during the journey to Mahopac. She had a pale, sickly color, her hands exhibiting large eschars of injuries she had inflicted upon herself during the fits. The temperature in the right limb and side of the face and neck was nearly one degree lower than on the other side — 82° Fahrenheit. Hands and feet cold and purple. Pupils rather dilated, but of equal size. Tongue coated at the centre, breath strongly offensive. She was very sensitive to touch. She could walk and run, carrying herself with the trunk bent forward. Careful inquiry made, did not detect paralysis of any kind. The girl was very talkative, prompt to reply,

and her mother, who gave the above information, stated that her memory was very good, although in other respects the mind was evidently impaired and her temper soured.

The urine of the night, examined the next morning after her arrival, was acid, of light color, density 1,021, without albumen or sugar, and loaded with triple phosphates. Pulse was very irregular, weak, and varying from 90 to 96. She was free from headache, and her bowels had not acted for some days.

Exclusive of some homœopathic treatment, the girl had never had anything done for her, and for three years past had manifested no change excepting in the summer of 1866, when she was for seven months free from paroxysms.

She was ordered :

Potass. Bromidi, - - - -	gr. xxx.
Ammon. Bromidi, - - -	gr. x.
Decoct. Calumbæ, - - - -	f. ℥ ss.—*Misce.*

To be taken three times a day, in addition to a pill night and morning with :

Ergotine, - - - - -	gr. ij.
Ext. Bellad., - - - -	gr. ½.
Pulvis Acaciæ, - - - -	q. s.— *Misce, ft. pil.*

She was to be packed every morning in the cold wet sheet, to have a very short shower bath towards evening, to exercise in light gymnastics, and to be put under a nutritious diet, with fat beef, claret, cream and coffee. She was also kneaded and rubbed every night and morning.

The first night she had several fits, and another slight paroxysm the next morning, after breakfast. She became irritable, and evading the vigilance of her nurse, ran away from the room where she was. The attacks repeated themselves through that night, and eight times again the next. The dose of bromide of potassium was then carried to forty grains every four hours. The convulsions ceased entirely from this time, but she continued complaining and crying whenever she was troubled ; the appetite failed more and more, until she would not eat anything : the breath turned excessively offensive, and a thick, whitish discharge ran from the mouth. This discharge existed before the bromide was used — though not so profusely, and was

diminished considerably by a solution of permanganate of potash used as a wash. The appetite, however, did not improve; she would not swallow any solid food, and on one occasion, the small pareels of beef given to her the evening before, came away the next morning upon washing out the mouth. She could not walk without staggering, or losing the equilibrium, and would scream if she were touched on any part of the body. If left alone she would bend down, to lie upon the floor. The mind, notwithstanding, continued unimpaired. Stimulants and nourishment by the mouth and rectum, failing to invigorate her, or to remove this condition, she died May 8, without exhibiting any new symptoms. The bromide of potassium was discontinued three weeks before her death, and cod liver oil given. The girl became comatose about twenty hours before expiring, and even until the beginning of this stage she would utter a cry as soon as touched in any part of the body. The pupils, naturally dilated, remained to the last responsive to light. The extremities were very cold and livid: upon the feet the epidermis was in some places raised by limpid serosity forming large flictenæ. The bowels did not act without injections, but there was no retention of urine. Finally, I may state that the diagnosis was entered as — Epilepsy connected with lesion of the cerebellum — probably a tumor.

I obtained permission from the mother to examine the head, and, aided by Dr. W. Royster, Assistant Physician to the institution, opened the skull fifty hours after death, the body having been kept in ice. The calvarium was so thin that in its upper part the diploë had entirely disappeared, making the bone quite transparent; the inner table easily separated from the dura-mater, which was very much congested. No adhesions between the membranes and the brain; the former was gorged with dark blood and distended by a limpid serosity, filling the sulci between the convolutions. Brain tissue very moist, firm, and highly congested, exhibiting, on section, a general sandy appearance. The same punctiform congestion found in the cerebellum, but the left hemisphere had undergone a degeneration involving the corpus dentatum, and giving to the parts a lardaceous resistance when cut through. The degeneration was in the main bloodless, irregular in outline, of a gelatinous yellow whitish tint, and limited to the centre of the cerebellum, without extending into the neighboring regions.

The oblong medulla and portion of the spinal cord to the level of the second cervical nerve, as also segments of the brain and cerebellum were saved for microscopical examination.

No membrane encysted the central lesion in the cerebellum, with microscopic characteristic of chollesteatoma, as shown in fig. 1, Pl. IX. Oval scales, and rectangular tables of cholesterine, mixed to amorphous matter interwoven with fine fibriles and nuclei of connective tissue composed this structure, surrounded by varicose and finely granular capillaries. I did not discover any granular corpuscles in this part, but fatty globules and granules. This degeneration did not reach the Pons Varolii; here was, however, observed the condition just described with the capillaries, besides an increased amount of amorphous matter, with fatty elements and amyloid corpuscles. The remarkable change existed in the spinal cord. The accompanying diagram shows a prepared section of the cord



at the origin of the second cervical nerve, as seen by myself and Dr. Royster. The cord, when fresh, exhibited a softened condition of the gray substance. Under the microscope the left anterior and posterior cornua, as well as the parts around the central canal,

were considerably destroyed. The structure had here a gelatinous aspect, stretched across by very fine brilliant fibriles of neuroglia, mixed with granular amorphous matter, and portions of capillaries irregularly distended and granular. Only the base of the posterior cornu had escaped such a complete destruction, and as to the anterior, it had nearly disappeared, while in this whole amorphous mass the cells could hardly be made out. It would have been interesting to ascertain the limits of this degeneration throughout the cord. The nerve fibres in the antero-lateral and posterior columns did not seem altered, excepting near the margins of the gray matter. This was equally but not uniformly involved in the medulla oblongata, and much damaged in its structure, particularly at the origin of the hypoglossus, with disfigured granular primitive fibres. The tissue of the brain and of both hemispheres of the cerebellum presented an uncommon quantity of free nuclei and meshes of transparent nucleated fibriles interspersed with albuminous granulations, also mixed with a transparent amorphous matter. The gray matter in the floor of the fourth ventricle was thickly stuffed with corpora amylacea, which were interspersed also in the medulla, and in the peduncles of the cerebellum, chiefly in the inferior ones.

The main features of this case so rapidly brought to a close suggest interesting comments. The staggering and want of equilibrium, the lack of strength in the back to keep an erect posture, and the extreme hyperæsthasia, were strongly indicative symptoms of the lesion met with in the cerebellum. But, with its left ganglion so deeply injured, and besides with such an extensive unilateral lesion of the gray substance corresponding with the above affected centre, no marked unilateral symptoms were observed, excepting the increased temperature on the left side and sweat-

ing on the right, both referable to derangement with the sympathetic.

The arrest of the spasms and the complete inability of the patient to recover, seem to be not casual coincidences but facts connected with each other. Evidently, when the girl entered the institution the disease manifested an active untoward march, the fits being more frequent, attended with notable impairment in the mental condition, and emaciation. No treatment had been opposed to this mischief, and it would seem that the bromide of potassium after controlling circulation in the nervous centres, arrested the chief source of convulsions without, however, having an influence over the alteration of these centres, already too far advanced to permit a renewal of their exhausted activity.

The most striking point in reference to the case concerns the state of the spinal cord, which brings further evidence against the theory regarding the transmission of sensitive impressions put forward by Brown Séquard. In this instance the central gray substance, that around the central canal, and both the anterior and posterior cornua in the left side, were deeply disorganized, and yet, sensibility was increased notwithstanding such an extreme damage to the channel through which it is transmitted. In a case of paralysis by J. Russell Reynolds, with pathological investigations by Lockhart Clark,* this distinguished

* British and Foreign Medico Chirurgical Review, April, 1864,

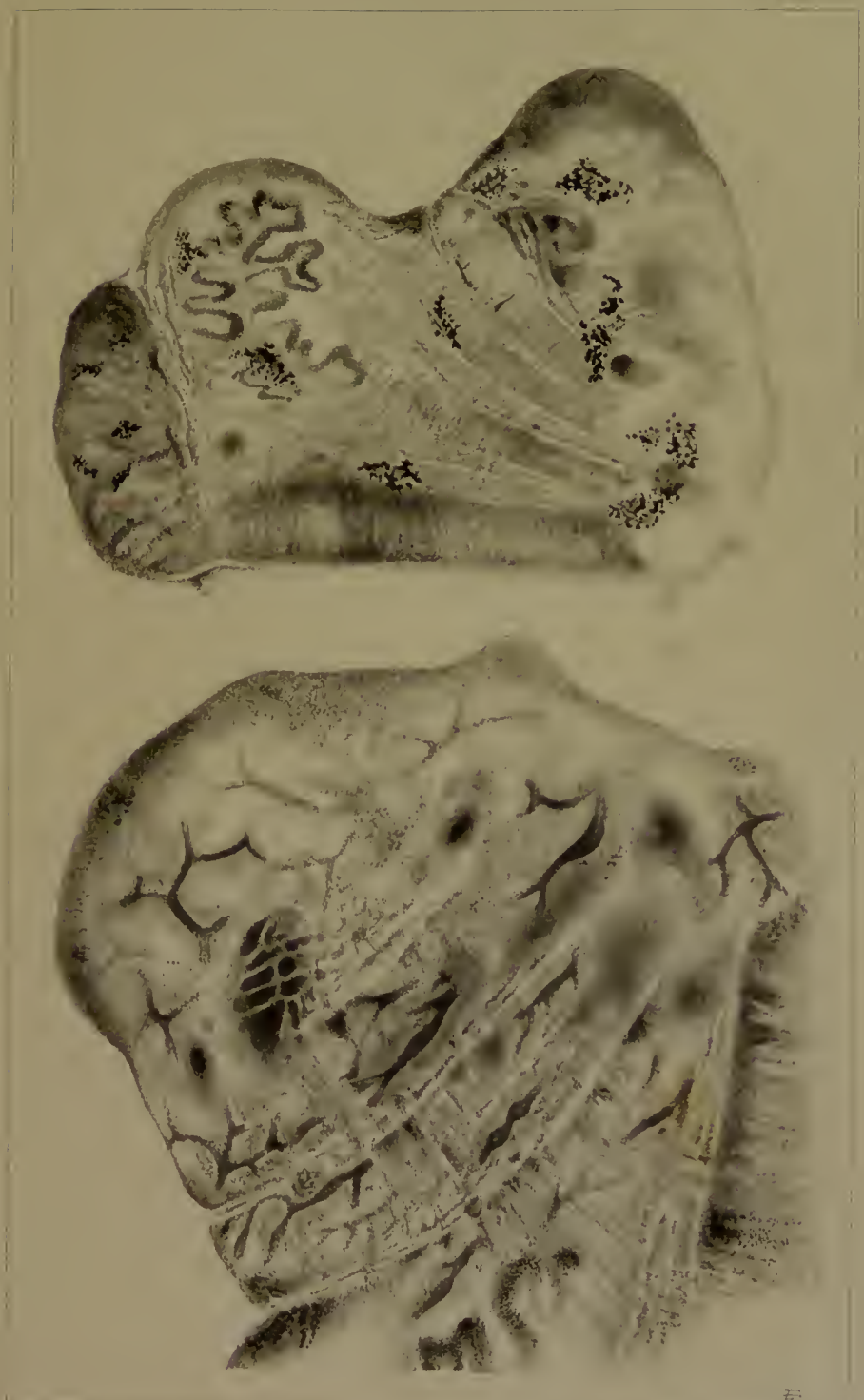
anatomist found that the central gray substance of the cord was destroyed on both sides, with painful hyperæthesia of the left arm, and not the slightest impairment of sensibility of the trunk and lower extremities. From this and other similar cases falling under his observation, Lockhart Clark has been led to reject the above theory. I have reported * a case of apoplexy of the spinal cord in which the gray substance had been in many places destroyed in its whole width without causing anæsthesia. Since that time, in addition to the case just related, I have met with a third remarkable instance, in which the gray substance of the cervical region had undergone an extensive fatty degeneration and sclerosis, without the patient having ever complained of anæsthesia. The history of this case, one of epilepsy, is given p. 124.

I need scarcely say that very few details remain to be added to those anticipated, about the pathological anatomy of the medulla oblongata, in the examples heretofore reported. The prominent changes of certain regions of the medulla in connection with the symptoms exhibited by the patients was first seized upon by Van der Kolk. Thus, with patients who did not bite the tongue, the origin and tract of the pneumogastric have been chiefly damaged, while with those who on the contrary did not bite the tongue during the attack, it has been the hypoglossal regions that suffered the greatest injury. This seems to be a very constant

* New York Medical Journal, June, 1865.

difference, regularly remarked by Van der Kolk, and which has been obvious in every case I have examined with an accurate knowledge of the symptoms presented by the patient. Under these circumstances, the aneurismal state of the capillaries, surpassing in size those of the remaining regions of the medulla, indicates as it were that they had been subject to a greater derangement. The appearance of such dilatations and a very granular condition of the walls in one of the capillaries from the nucleus of the pneumogastric in Case X. is represented in fig. 2, Pl. VII. In this occasion the blood vessels, at the nucleus and along the path of the nerve, had from three to four-tenths of a millimetre in a diameter, whereas they hardly reached this dimension in the rest of the medulla, where the largest, including those of the hypoglossus, were not more than from two to three-tenths of a millimetre wide. The coats of this vessel were to such a degree encrusted with fine granulations, that in some points their thickness reached three and four-hundreths of a millimetre. In Case VI. a minute artery in the tract of the hypoglossus had been distended to the extent of a forty-fifth of a millimetre; but the aneurismal dilatation proceeded from blood mixed to fatty molecules and white cells, extravasated through a rent in the tunics of the vessel, and therefore solely distending the adventitious sheath.

All these morbid changes of the medulla oblongata, precisely similar to those of the brain and cerebellum, though important, do not, indeed, prove that epilepsy



MEDULLA OBLONGATA EPILEPSY

is more attributable to them than to the lesion in the latter nervous centres. I have previously advanced that in no instance of epilepsy have I met with degeneration of the brain or cerebellum unaccompanied by that of the medulla, and I will presently detail instances of confirmed epilepsy, in which on the contrary every region of the encephalon, excepting the medulla, was free from alteration appreciable to the naked eye, or under the microscope. I lay special stress on this phenomenon as it seems to be not accidental, for it has been brought under my notice four times out of the twenty-six post-mortem examinations I have made of epilepsy, and in three more occasions the cerebellum participated in a slight degree of the degeneration of the medulla, no trace whatever of it being detected in the brain.

CASE X. *Epilepsy. Death from double pneumonia. Hepatization of the lungs. Brain and cerebellum unhurt, medulla oblongata and sympathetic degenerated.*

In September, 1866, a boy three years old, died at the infant's department of the Alms House. He had been subject to epileptic fits, occurring while teething and thereafter repeating regularly since the convalescence of measles, with which he was seized the spring before. He was a bright looking child, with head well developed, and came to his end upon sudden attack of double pneumonia, few days after a series of severe fits. I saw this child in two of the paroxysms; they were of a most violent nature, ushered in by a cry, with great lividity of the face, general convulsions, a good deal of frothing at the mouth, without biting the tongue and leaving the little patient very much exhausted.

Autopsy.— Subpleural ecchymoses in the posterior surfaces of the lungs, in a state of mingled red and gray hepatization, with emphysema in the anterior part of the upper lobes. Heart healthy with

right cavities filled by coagulated blood. Nothing abnormal about the abdominal viscera. Skull with ossified fontanelles. Membranes congested; sinuses of the brain full of fluid blood; small amount of serosity effused beneath the arachnoid. On different sections of the brain and cerebellum the tissue appeared without any unnatural modification; nor did the most careful microscopical search discover any morbid state of their minute structure. The capillaries, chiefly in the convolutional gray substance, still maintained the irregular shape and granulated transparent walls with scattered nuclei, proper to early infancy, which I had noticed before in specimens prepared by Professor Robin, for his private courses, and in my subsequent pathological investigations of the brain of children. This transient hystological condition of the cerebral capillaries during infancy, has been recently and particularly pointed out by Hayem and Laborde.*

The medulla oblongata showed definite and extensive evidences of morbid change. Everywhere, and indeed in every transverse section, were discovered the different stages of a granular albuminous exudation, in many places interspersed with corpora amylacea, figure 2, Plate VIII, which were abundant beside in the ependyma of the fourth ventricle. The neuroglia soft and fragile, displayed several nuclei in its finely reticulated structure. The nerve fibres, preserving their form, but granular, were easily disintegrated, as also the cells, many of which, in the posterior regions of the cord and in the nucleus of the pneumogastric, appeared likewise granular and filled by a dark-brown pigment, imparting a peculiar tint to the specimen when examined by reflected light. This condition of the cells was like that detected in those of the cervical sympathetic, figure 1, Plate IX. At the olivary bodies the infiltration of pigment nearly obliterated the corpus dentatum, replaced by a dark-colored mass. The dilatation of the blood-vessels was extreme in the nucleus and course of the pneumogastric, figure 2, Plate VII. An amorphous, granular exudation, intermingled with corpora amylacea surrounded the vessels. The primitive fibres of the vagus, in large part reduced to the cylinder axis and sheath, with a gray transparent color, and elongated nuclei, were separated by interstitial heaps of translucent

* Op. cit., p. 217.

PLATE VII



A Laboratory

AMYLOID DEGENERATION OF THE MEDULLA IN EPILEPSY

Edinburgh: Oliver & Boyd, Printers, Co. 98 W. 75th St. N.Y.

fibres with distinct nuclei. Nowhere in the origin or path of the hypoglossus did the aneurismal dilatations equal those of the blood-vessels connected with the pneumogastric. They reached in the vicinity of this nerve thirty-seven hundreths of a millimetre, whereas in the regions of the hypoglossus they did not, for long together, go beyond twenty-eight hundreths of a millimetre.

In the cervical sympathetic the cells, as already said, in a granular state had their nuclei masked by the abundance of pigment granules. Transparent fibres with large granulated nuclei pervaded the whole ganglia, and the primitive delicate nerve tubes, with nucleated sheaths, had undergone a dark granular change. Figure 1, Plate IX.

This case, therefore, seems to be very strong evidence of the essentially primitive part that the interstitial morbid process of the medulla oblongata takes in the production of epilepsy. Many of the specimens here alluded to, were examined also by Dr. J. B. Done, my friend and colleague at the Charity Hospital. Figure 2, Plate VII, represents a section of the cord at the origin of the vagus and hypoglossus.

CASE XI. *Epilepsy from fright. Pulmonary tuberculosis. Death. Brain and cerebellum normal, medulla and sympathetic degenerated.*

Margaret O'B..... æt. 22, became epileptic at the age of nine, in consequence of fright. Fits discontinued three years after to recur on the establishment of menstruation, at the age of fourteen, repeating with greater and greater severity at every menstrual period. Used to bite the tongue during the spasms and urine was then involuntarily voided. She died in the Alms House, Charity Hospital, with tuberculous consumption.

Autopsy.—Pleuritic adhesions over both lungs, with gray tuberculous infiltration, and small suppurated cavity at the apex of the right lung. Heart flabby but normal, uterus nulliparous, enlarged, with superficial abrasion of the neck, plugged by a transparent thick mucus. Ovaries with corpus luteum recently formed in the left,

and small cysts in the fimbria of the Fallopian tube. Cerebral membranes bloodless, no serous effusion of any account in the meninges. Cavity of the ventricles with about one ounce of limpid fluid. Choroid plexus pale. Brain, cerebellum, medulla oblongata, cervical sympathetic, and solar and lumbar ganglia, prepared for microscopical examination.

In this, as in the former case, not the least impairment could be made out in the structure of the brain and cerebellum. The medulla oblongata, and principally the olivary bodies, exhibited a typical degeneration involving the nucleus of the hypoglossus and that of the vagus. The cells on the inner side of the hypoglossal roots, corresponding to the great pyramidal nucleus of Stilling, the posterior cell groups connected with the vagal and hypoglossal nuclei, and those of the post pyramidal one, were much disintegrated and infiltrated by pigment, fig 1, Pl. v. Delicate fibres of neuroglia, with granulated nuclei, entangled with fine amorphous matter, and dark molecules, prevailed between the nervous elements. Corpora amylacea were detected only on the floor and in front of the apex of the fourth ventricle, and in the tracts of the hypoglossal nuclei. The difference between the capillaries in these regions, and those of the brain and cerebellum was very conspicuous. In the medulla they were over distended, various and embedded into a sort of translucent granular matter, containing many granular corpuscles, which seemed connective nuclei enveloped by surrounding molecules. The dilatation of the blood vessels, not exclusive to the regions of the hypoglossus, could be further made out in the vicinity of those cells connected with the vagus and along its roots.

The cervical sympathetic cells, without nuclei and reduced to a mass of opaque granulations, seemed extinguished by the hypergenesis of transparent connective elements, and amorphous matter. The capillaries around the ganglia had also undergone a granular degeneration and dilatation rendering them very friable. They contained blood with more white cells than corpuscles, and distinct fatty molecules. This same structure could be noticed in the solar and lumber ganglia; but with them the preponderant connective tissue was interspersed with corpora amylacea. The contents of the primitive fibres connected with these ganglia had either disappeared or taken a strange gelatinous appearance, which I had not seen before.


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CASE XII. *Epilepsy. Death from diarrhœa. Brain and cerebellum normal. Amyloid degeneration of the medulla oblongata.*

This was a foundling girl, over a year old, of the Infant's Department, Charity Hospital, who died with diarrhœa. She had epileptic fits preceded by yawning, or sudden starts, and frequently occurring through the day, for several months before her death. Microscopical examination of the brain and cerebellum did not disclose any departure from normal structure, but the posterior regions of the oblong medulla had evidently suffered the above described degeneration, with increased amount of connective nuclei and corpora amylacea. The sympathetic was equally injured, and the abdominal ganglia presented a considerable quantity of pigment giving them a peculiar brick color.

CASE XIII. *Epilepsy with dysphagia and dyspnœa. Pulmonary tuberculosis. Apoplectic effusions in the medulla oblongata without any other lesion in the rest of the encephalon.*

In 1864, I attended a young man, aged 24, who came under my care through the dispensary of the St. Catharine's House of Mercy. He had experienced no severe illness in his life time, but for the prior year and a half had been troubled with epileptic fits, beginning directly after an attempt to strangulate himself in a moment of despondency. I saw this patient frequently. His hands were cold but not the feet. The pupils usually dilated. For an hour after the attacks he experienced difficulty in swallowing or taking any satisfactory inspiration. He did not feel comatose after the paroxysms, which occurred once or twice weekly, at times with momentary vertigo, and always apt to be present upon any irregularity of the bowels. Symptoms of consumption rapidly developed themselves, and the patient becoming extremely emaciated, died two days after the last fit, the 21 Oct., 1864.

I obtained permission to examine his head. No external evidence of lesion could be detected in the brain or cerebellum, nor did I discover it under the microscope. The seat of injury lay altogether in the medulla oblongata. Here, in the centre of the right olivary body, and in the marginal regions of the left, there appeared

speckled effusions from capillary vessels, very much distended in other places, as in figure 1, Pl. VII. The pneumogastric and hypoglossal cells, granular and broken up, in the midst of hæmatic crystals and granulations, were deeply injured on the right side. On the left, the extravasations less numerous, were principally displayed in the centre of the corpus dentatum and the post pyramidal nucleus. The capillary aneurisms could be discovered in the upper limits of the cervical region, the tissue throughout the medulla and cord being irregularly invaded by exudations of a fine amorphous matter.

The spinal gray matter, uninjured in the centre, exhibited a gelatinous granulated appearance, less advanced in the anterior than in the posterior cornua. In the columns, the friable nerve-fibres preserved their shape, while the intervening neuroglia had very much increased.

The trouble with deglutition and inspiration, experienced by the patient after the attacks, are plainly accounted for by the lesions at the origin of the hypoglossus and vagus, which through the phrenic nerve, acted on the diaphragm. As to the rapid development of phthisis, it probably depended on the lesion of the medulla.

The cases just laid before the reader, irrespective of those in which the cerebellum suffered slight injury, render evident that the medulla oblongata is the primitive seat of epilepsy, which is capable, therefore, of existing without any organic modification of the brain. But, could not these be regarded as exceptional instances? I think not; and further experience will undoubtedly increase the proportion of such cases. Nevertheless, the propagation of the disease usually reaches the most distant limits from its inception, or the attacks may as frequently be superinduced upon consecutive degeneration of the medulla, as I have distinctly ascertained on cases of epileptiform convulsions.

Bouchard says, that: "Epileptiform and marked epileptic attacks are frequently observed in subjects afflicted from infancy with hemiplegia, and not infrequently in old persons affected with cerebral softening, and in whom, we find at the autopsy considerable atrophy of one peduncle of the protuberance and of the medulla. I must acknowledge that we are not as yet capable of positively verifying this hypothesis; but it seems to me probable, because the epileptic attacks were very intense, and very frequent in a patient exhibiting a high degree of sclerosis of the medulla, from compression of this organ, and because we, occasionally, see the contraction notably increase in the paralyzed limb, a few moments before the epileptic paroxysm."* I need not repeat that, I have verified the correctness of the views put forward by Bouchard.

According to Virchow,† "an anatomical or physiological unity has at least as yet been nowhere demonstrable. If we really were to set down the nervous system with its numerous separate centres as the central points of all organic actions, even then the thing actually sought for — a real unity — would not have been obtained." An obvious correspondence exists between the phenomena of normal action and those of pathology or morbid physiology. The impos-

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* Des Degénérations Secondaires de la Moelle Epinière. Arch. Gén. de Méd. Paris, Sept., 1866, p. 295.

† Cellular Pathology, p. 323.

sibility of an anatomical unity makes, therefore, the brain and medulla subject to the material changes originally induced by epilepsy, and for this very reason the cause may still extend its effects to and over distant though not unconnected regions of the cerebro-spinal system. No physiological function is discharged without a material organic modification, no morbid process or disease whatever, can be originated, unless some organic derangement takes place. The conclusion that must necessarily be drawn from this fundamental fact, is, that if maladies might arise from immaterial disturbances without any organic seat, they would never display such variety in different individuals. It is at present held that, inasmuch as epilepsy may supervene upon any possible cause, with every region of the nervous system, whether damaged, or on the contrary completely sound, we may logically deduce that no localization of the disease can be attempted. But, what thus appears so well established is untenable if we only reflect that such an assumption is grounded upon speculative and incorrect views, because there is as yet, so far as I know, not a case on record in which the above sound condition is substantiated by a thorough microscopical investigation of the encephalon. We can no longer place reliance on the external appearances of the organs; the reform effected on our notions of tetanus, evinces that they are not altogether trustworthy. Although the spinal cord has been examined without any change in its tissue, being detected, it nevertheless,

displays, in cases of tetanus, numerous lesions and alterations of structure, appreciable only under glasses of high magnifying power, as demonstrated lately by the researches of Lockhart Clark. And this is an example which holds good for the pathology of many other neuroses.

The cardinal fact so wisely comprehended by Marshall Hall, and warranted by the pathological researches of Van der Kolk, only needed, in order to be completely ratified, the evidence of instances in which epilepsy had long existed with its typical symptoms and no other but the primary lesion of the medulla oblongata. This demonstration we get in the above cases. The last one is a true reproduction of the experiment so skillfully performed by Brown Séquard, and the accordance between them could not be more complete to establish the part accomplished by the medulla oblongata. Moreover, clinically we meet with as unequivocal evidence of this essential connection of epilepsy with lesion of the medulla without participation of the brain. I have actually under my care a girl 15 years old, the daughter of a gentleman residing in Washington, whose case is a remarkable instance bearing on the point in question. She was thrown out of a sleigh, four years ago, fell on her right arm, bruised it near the elbow, and became instantly seized with cramp of the right limbs. The attacks recurred a year after, and since then have been frequently repeated extending over the muscles of the right side of the trunk. The paroxysm always com-

mences with a peculiar tingling sensation and contraction in the ring and little fingers, creeping up the arm to the spine, thence traveling to the leg, until the whole side is convulsed. The right arm and hand have grown less than the left, the difference being hardly perceptible between the feet. The right upper limb is also colder, and its skin drier than the opposite. The attacks have been attended with unconsciousness but once and that lately; they occur during the day or night, and have sometimes been repeated as many as fifteen or twenty-five times in succession. Running, when she is warned by the aura, checks considerably or prevents the attacks. When seized, she becomes at first pale, with pupils largely dilated, and utters a cry, as the back and limbs get violently convulsed for a second or two, without losing her consciousness. She does not go to sleep upon the attack, nor bite the tongue during it, and she comes out of it very weak and depressed. The application of a moderately induced current to the ulnar nerve, or to the lower cervical region, has on two occasions induced a paroxysm. I have not been able to ascertain whether a ligature around the wrist will prevent the fit. Cauterization with a red heated iron of the lower cervical region, has determined a change in the temperature, with more comfortable feeling of the arm. The burning is to be renewed along with gymnastics, hydropathic and other proper means to increase the nutrition of the limbs. Bromide of potassium, and of ammonia, have been unsuccessfully tried by the patient before I saw her. In addi-

tion to the above means, she is using conium with ergotine, and arseniate of potash. I look upon the case as one of *neuritis propagata*, the medulla oblongata becoming involved upon prolonged incident irritation. I may also state, that when the girl first saw me, she had a scattered vesicular eruption and a boil on the right forearm, which I attributed to the peripheral nervous lesion. Evidently, this is a characteristic instance of spinal epilepsy.

Among the various lesions of which I have treated, those of the sympathetic have been generally mentioned. I should judge further comment on them superfluous either to point out their nature, already defined, or their obvious relations to the changes on the medulla oblongata. Granular degeneration, irregularity, and pigment infiltration of the cells, in addition to more or less hyperplasia of connective elements, have prevailed in the sympathetic, reflecting as it were the influence of the disturbance giving rise to the modifications in the other nervous centres. I am inclined to consider such as a primary and not as a consecutive derangement, for it is not less the fact that it has appeared in every instance and more constantly than the cerebral alterations. The propagation from the fourth to the third ventricle, corpora striata, and cortical substance, frequently noticed, would seem to follow up the tract of sympathetic fibres in the brain, which may be perhaps the first to be disturbed in this organ. It still remains for me to assert that I have examined the sympathetic, namely,

in fifteen cases of epilepsy, without failing, as already stated, to detect a more or less impaired state of the cervical ganglia. Not unfrequently, there has been a conspicuous similarity between the injured ganglionic cells and those of the medulla, or in the middle and between the cornua of the spinal gray matter. But, although, as established by Jacubowitsch, sympathetic cells are located in these regions, yet, I have not sufficient evidence to ground the opinion that the sympathetic suffered more damage than any other cells, or actually, that those in the spinal cord, so hurt, were mainly sympathetic cells. Neither have I arrived at distinct results every time, on investigating the net work or nervous plexus around the cerebral arteries, participating in advanced cases of the degeneration in the arterial parietes, and displaying proliferation of nuclei in their attenuate primitive fibres.

There is, in cases of epilepsy attended with paralysis, a phenomenon, *i. e.*, immediate absence of cadaveric rigidity and earlier putrefaction of the paralyzed muscles, which I attribute to the lesion of the sympathetic. The fact is principally exhibited by the muscles of the paralyzed limb which are affected with permanent contraction. I have before pointed out this curious phenomenon in cases of reflex paralysis,* and similar observation has been made by Charcot and Bouchard.†

* On Reflex Paralysis, its Pathological Anatomy and Relation to the Sympathetic. New York, 1866, pp. 30, and 79.

† Loco cit., p. 295.

I must not overlook the change discovered in the ganglionary roots and peripheral extremities of the dorsal nerves in a case I shall have occasion to refer to hereafter, and in which an herpetic eruption of zoster encircled the base of the breast. The ganglia presented their cells reduced to a mass of pigment deposited in the granular contents, with an exuberance of connective tissue and nuclei, which did not exist so much in the other regions. As to the nerves connected with the eruptive patches, they had undergone a fatty degeneration of the primitive fibres, with the same rank growth of connective elements. The blood vessels in the vicinity of these spots exhibited a marked amyloid degeneration. This is not, however, the only example I have met with of such structural change. I have noticed it four times more, without the amyloid condition of the blood vessels, on investigating the ganglia and peripheral nerves, in relation to similar eruptions and modifications of the skin commonly observed with epileptics, and I have likewise found them in old standing cases of paralysis with ulceration of the lower extremities. These phenomena are in accordance with those pointed out by Recklinghausen and Baerensprung,* in the case of an infant with unilateral zona on the breast; more recently, Charcot and Cotard,† have likewise reported a case of

* Beitræge zur Kenntniss des Zostes, XI Bd. 2e Heft, p. 90, 1863, in Annalen des Charite Krankenhauses zu Berlin.

† Mém. de la Société de Biologie, 1866, p. 41.

alteration (neuritis) of the right cervical plexus and corresponding spinal ganglionic roots, attended with zona on the same side of the neck. In this instance the ganglionic cells were equally infiltrated with pigment on both sides, but the vascularity was increased on the right ganglia with hypergenesis of connective tissue and nuclei extending to the neurilema, the primitive fibres, however, preserving their normal character. I believe that these and other disturbances, to be presently pointed out, in reference to the capillaries, are entirely caused by the sympathetic. Vulpian,* and prior to him Koëlliker,† admit that — there are nerve-fibres directly arising from the ganglionic globules. "These ganglionic fibres, proceed, in by far the greater part, perhaps all, in a peripheral direction; they join and reinforce the root-fibres, passing through the ganglion, so that every ganglion is, accordingly, to be regarded as a source of new nerve-fibres." It is true, that independently of the ganglion, injury or irritation in the course of the nerve may determine the same abnormality of the skin, as in cases reported by Paget, Charcot, Rouget, Mitchel, Morehouse and Keen, and by Gerhart, who has twice seen the application of a constant current to the mental branch of the inferior dental nerve after its exit from the mental foramen, attended with a vesicular eruption on the chin.‡

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* Journal de la Physiologie, etc., 1862, p. 32.

† Manual of Human Microscopic Anatomy. London, 1860, p. 244.

‡ Central Blatt für Die Medic. Wissench, 1866, No. 4.

But it is easily seen how, in every one of these instances, the interrupted central trophic influence of the ganglia originated such derangement in the peripheral vegetative process.

I will not pass in silence the condition of the cutaneous nervous filaments in the case of a young man subject to epileptic fits, commonly occurring in the morning at the moment the urine, as it was voided, touched the orifice of the elongated prepuce. The patient was addicted to masturbation, the determining cause of his fits. Upon consultation with my friend Professor Wm. H. Van Buren, we concluded to remove the sensitive portion of the prepuce, and he accordingly performed the operation for phymosis. I examined the nerves of the cut off integuments and found, that the primitive fibres, contained in a dense layer of small nuclei and fibres of connective tissue, were stretched, with their contents either reduced to the cylinder axis or to the myeline irregularly accumulated in granular fragments. Such a hypertrophy of the neurilema resembled that described by Verneuil,* although in this instance of mine, the patient did not complain of pain in the prepuce, which existed very acutely in the case of Verneuil, who proposes the name of cylindro-plexiform neuroma (*nevrome cylindrique plexiforme*) for this peculiar degeneration.

The circulatory system is especially concerned in the pathogeny of epilepsy, as may be seen in the fore-

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* Archives Gén. de Médécine 1861, Tome II, p. 537.

going cases. There appears from the onset of the epileptic disease, a disturbed action, and, whether due to the slowness or complete stoppage in the supply of blood, or to a primary trouble in the nervous elements, it soon brings a structural modification of the blood-vessels. And it is not less positive, that the metamorphosis commences with paralysis and dilatation and closes with a retrograde or fatty degeneration of the blood vessel. I assert that, so far as I have had opportunities to judge, the dilatation is mainly the result of paralysis and not of partial contraction in the calibre of the blood vessel, on account of the lengthening associated with it, which could proceed only from a lack of tonicity in the vascular walls. Even when a thrombus or migratory clot obliterates an artery causing stagnation of blood, the aneurismal dilatation acknowledges as cause, in great part, the weakening of the vascular walls. However, in this latter circumstance, there are two more important elements to take into account: the increased tension of blood,* in consequence of the obliteration of the vessel, and the reflux of venous blood from want of vis à tergo in the artery, as imagined by Virchow. To the impediment in the local circulation must be referred the circumscribed abnormities in the structure of the nervous centres, and the thickening, or growth of granulations and adventitious patches, in the cerebro-spinal

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*See Marey, Physiologie Médicale de la Circulation du Sang, Paris, 1863, p. 146, and Prevost and Cotard, op. cit., p. 40.

membranes. These adventitious deposits do not belong, as generally supposed, exclusively to old age. I have not seldom encountered them in individuals dying in the prime of life from epilepsy or other cerebro-spinal affections.

In a most elaborate paper on loss of speech and its association with valvular disease of the heart, and with hemiplegia on the right side, Dr. Hughlins Jackson, of London, deals with the subject of unilateral epileptiform convulsions, occasionally present, and which he, from the most general characters, ascribes to disease in the regions of the middle cerebral artery. In this history Dr. Jackson * keeps almost entirely to the clinical aspect, and says little or nothing about the exact convolution of the brain, damage of which produces loss or defect of speech. He approaches the possibility of embolism through the evidence of the cardiac disease, together with the occurrence of hemiplegia and aphasia, with or without epileptiform convulsions, being, further, led to advocate the theory of Broca, that: disease of the left side of the brain only, produces loss of language; and, moreover, that the faculty of articulate language is located in a very limited part of that hemisphere, namely, the posterior part of the third left frontal convolution. It would be inconsistent with the plan of this work to dwell at any length on the still vexata question of aphasia. Although the proofs brought forward by

* London Hospital Reports, vol. 1, 1864, p. 389.

those who uphold the doctrine of Broca appear very conclusive, for my own part I can not assent to the exclusive solution they give to the question, and doubt very much if any full difference of function, between the hemispheres of such a symmetrical organism as the brain, exists. The development of the cerebral convolutions is undoubtedly subordinate to the performance of the intellectual processes, but I have often thought that such puzzling arrangement of the superficial strata is, perhaps, only subservient to the great activity of circulation demanded by the incessant generation of force accomplished by the brain, and that, consequently, the disposition has been wisely intended not to limit territorial regions for special faculties, but to afford a wider range for more uniform and regular circulation throughout the cerebral tissue. I look upon the ganglia in the brain and medulla as the main centres of our faculties, more or less directly connected with the peripheral layers of the encephalon through tracts not as yet exactly determined. In the absence of other facts, their genesis, prior to that of the convolutional gray matter, further indicates that they must be chiefly endowed with the discharge of important functions. And, as William Turner states, it "should be kept in mind that a convolution, though separated superficially from its neighbors by fissures, yet becomes continuous with them at the bottom of these fissures, so that the gray matter of the one blends with that of another. Again, adjacent gyri, though in many respects they may present precise morphological positions

and relations, are not unfrequently connected together by secondary gyri, either altogether superficial or partially concealed within the dividing fissure, so that we can not with them, as with a group of muscles, make a subdivision into distinct organs without effecting an artificial separation of their contents."* Moreover, it is not unworthy of note that, superficial lesions of the brain engender any trouble when they derange consecutively the basal ganglia, with which they are connected, and daily experience demonstrates that the most extensive superficial injury of the brain may thus exist in a latent form. Be this as it may, the arguments against the views of Broca, have been ably discussed by those who reject them, and who, in addition, have presented instances of aphasia without injury of the third frontal convolution, and yet, with lesion of other distant cerebral regions. Indeed, Broca shows in every one of his examples the undoubted association of aphasia with lesion of the region in question; but this is only one part of the problem. Will this prove that such left convolution is exclusively deranged in aphasia, and that other cerebral regions be not as efficiently involved? Will this explain the no less positive occurrence of lesion of the left anterior lobe, without aphasia; or of the Pons Varolii alone with loss of speech, as I have twice had occasion to see? It will be too much to affirm any negative conclusions,

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* The Convolutions of the Human Cerebrum Topographically Considered. Edinburgh, 1866, p. 28.

but with the knowledge of such facts staring us in the face, it is not too much to say that they have almost the force of demonstration to the improbable absolute correctness claimed by such doctrine. I am fully convinced, after the most zealous scrutiny, that lesion of the left third frontal convolution fails to be constantly attended by symptoms of disturbed faculty of speech, but it is perhaps best not to insist now on this absence of aphasia, and I will, therefore, limit myself to the subject of disease in the arterial regions of the brain associated with epilepsy and aphasia, standing for my assumptions on evidence furnished by my own post-mortem examinations.

Dr. Jackson, who undoubtedly has been the first to point out or establish the coincidence of aphasia with cardiac disease, has brought out, in the paper referred to, only one instance, Case XIX.* of epilepsy, wherein "there was a loud diastolic murmur heard best at the ensiform cartilage." He further adds: "However, it still remains that I have never yet had under my observation a patient who had, when I saw him, notable defect of speech, with hemiplegia on the left side, for this patient conversed well when he came to me, but he articulated badly. Yet, it was a kind of talking which I believe follows loss or defect of articulate language, and not mere difficulty from want of power in the lips, tongue or palate." In this case, which was a remarkable one, for the hemiplegia and

* Loc. cit., p. 438.

subsequent epileptiform seizures were on the left side, the patient became speechless, when first struck with hemiplegia, and for six weeks did not speak at all, except to say "yes" and "no," and then asked what time it was. About five months after the first attack a second came on, but this was not followed by any notable paralysis. It was a convulsive seizure, and affected one side only, the one previously paralyzed. It began in the left cheek, and next the arm and leg were convulsed, and the patient became insensible. In a foot note to the case, Dr. Jackson says, that on referring to a letter of Mr. Corner, who sent the patient to him, he found that at the time of the first attack there was "loss of power on left side, so far as limbs are concerned, but on the opposite side of the face," and thereupon, Dr. Jackson assumes that it is fair to conclude that there was at first disease on each side of the brain. Of the remaining five cases of unilateral epilepsy, in one, Case XIV, there was no valvular disease, but irregularity of the action of the heart. In Case XXIII, we find: loss of speech and hemiplegia on the right side — Epileptiform seizures; no notes as to cardiac disease; death from apoplexy. Dr. Jackson* says: "I regret that all the information I have about the autopsy, except that there was effusion of blood all over the surface of the brain, was, that one of the anterior pyramids, and I think one of the corpora olivaria was smaller than the other. . . . I am

* Loc. cit., p 445.

sorry that I did not examine the vessels at the base more carefully. I can only surmise that there was an aneurism of the left middle cerebral artery, and that the loss of speech and hemiplegia resulted from the occlusion of some branches of this vessel, and that death occurred from rupture of the aneurism." In Case XXVI, convulsions left the patient hemiplegic on the right side, with loss of speech. Of the remaining patients, one, Case XXXVI, had complete loss of speech, gradually coming after bilateral chorea. Convulsions on right side; no evidence of cardiac disease. Finally, Case XXXVI, is an instance of loss of speech and hemiplegia on right side, followed by unilateral convulsions beginning by a tingling in the right hand, the result of a blow, causing "a depression of the skull, about a square inch, on the left side of the middle line, about the anterior third of the parietal bone."

From the preceding abstract it may be seen that, in one single instance was the cardiac disease distinctly made out, and also that the only post-mortem examination reported leaves the case dubious, inasmuch as the atrophy of one of the anterior pyramids and one of the corpora olivaria points out complicated lesions in the brain and medulla oblongata.

As to Case XIX, it is very plain that the affected side of the brain must have been the right. Facial paralysis is usually not a transient accompaniment of hemiplegia, and if it did acknowledge the same cause of aphasia, it is natural to presume that it would have been likely to reappear with the second attack, when

the left cheek became convulsed. But, without resorting to any speculations on the subject, the occurrence of crossed hemiplegia, *Hémiplégie alterne* of Gubler, is a proven fact, and it proceeds from lesion of the Pons Varolii, which probably existed in this instance.* I will add on behalf of this assumption, that among the records of defect or loss of speech without evident microscopical lesion of the third left frontal convolution, I find two in which the symptoms attended a lesion of the Pons Varolii. Before briefly reporting them, I must say that Bouchard has also recorded one case of aphasia, without lesion of the third left frontal convolution, and with yellow softening of left corpus striatum and secondary atrophy of the Pons Varolii.†

An old woman died at the Hospital for Paralytics, with left facial paralysis, loss of power in the right limbs, and impossibility of articulating or employing certain words. The middle and posterior lower part of the Pons, close to the peduncles of the brain, exhibited a white softened patch the size and shape of a bean. Left corpus striatum congested and softened. No change detected in the third left frontal convolution.

A man, in the same division for paralytics, dies with external strabismus and facial paralysis on the right side, and paralysis on the limbs of the oppo-

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* This has been written when I found that Dr. W. J. Ogle entertains the same views as to this case, which he considers one of alternate paralysis.— *On Aphasia and Agraphia*, St. George's Hospital Reports, vol. II, p. 119.

† Mém. de la Société de Biologie, 1864, tome I, 4ème série, p. 111.

site side. He could speak only "yes" or "no," though he understood what he was asked. A small surface, extending from the origin of the crus cerebelli to the floor of the fourth ventricle, and the right olivary body, were in an advanced stage of sclerosis, also present in the corpora striata and optic thalami. No such degeneration existed in the left third frontal convolution.

Aphasia existed in only one of my own preceding cases, but with deep degeneration of both anterior lobes of the brain. Strongly contrasting with this example I will now report another of sclerosis of the gray substance in both third anterior convolutions, without any aphasia. In Cases III and IV, the condition of the third anterior convolution was carefully investigated and found evidently injured; yet, neither patient exhibited any loss or defect of speech, as ascertained by myself and the Assistant Physicians. It is true, that in Case III, the patient was reluctant to talk, but she nevertheless could give us, to the last, an account of her condition, without ever indicating any aphasic trouble. I now append two remarkable cases akin to these latter, extracted from records kept by Dr. Castle. The drawing, figure 1, Pl. VIII, is from a photograph of the specimen.

CASE XIV. *Epilepsy. Dysphagia and Paralysis of the tongue. No aphasia. Sudden death from hæmatorrachis. Double aneurism of the Vertebral arteries. Sclerosis of the brain, medulla oblongata and spinal cord. Degeneration of the ganglia and nerves connected with a herpetic eruption of the chest.*

Denis D . . . . , aged 67 years, native of Cork, Ireland; occupation, clerk; died at the Hospital on the night of May 24th, 1866, while

straining at stool, a small quantity of blood coming from his mouth. He was from his youth subject to epileptic fits, and for months before his death noticed a feeling or dizziness of the head upon attempting to walk, with difficulty to move or to protrude the tongue. The fits had decreased in frequency, but his condition growing worse on account of incapacity to move, he was obliged to keep in bed. His sleep was broken, the decubitus dorsal; expression of the face dull; pupils contracted readily; muco-purulent discharge from the conjunctivæ. Tongue clear and, though with difficulty, protruding in a direct line; palate flabby. Temperature of the skin normal. Anorexia. Bowels rather loose, no tenderness of abdomen. Respiration slow. Pulse eighty, irregular, and small. Heart enlarged, with rough aortic direct murmur. Urine normal. Has never had syphilis, but gonorrhœa three or four times. There is an eruption of herpes zoster, like a girdle, around the base of the breast. The mind to the time of death was clear, though at moments clouded by a kind of confusion or absence in his ideas. At other times he would lose the power of articulating and had to whisper words with a considerable effort. This was accompanied with dysphagia. Otherwise he could talk without impediment and express himself with ease about his ailment. As above stated, he was unable to move from partial paralysis of the limbs, which were stiff and rigid. He also complained of pain with occasional cramps, or jerking, of the lower extremities. No impairment of sensation excepting numbness in the feet; sensory impressions seemed, however, distinctly perceived. I visited him the afternoon before his death, he did not show then signs of aphasia.

Autopsy.— Heart enlarged, atheromatous deposits in the aortic valves. Arch of the aorta extensively changed, with large calcareous and atheromatous patches. The other viscera displayed nothing remarkable. On removing the scalp and membranes, the brain appeared dry, and bloodless in its upper surface. At the base, the Pons Varolii was covered by a film of coagulated blood, extending into the spinal canal, and the medulla and cord embedded in a mass of grumous blood, proceeding from the rupture of an aneurism of the right vertebral artery, near its junction with the left to form the basilar trunk. The left artery was equally dilated: a clot, plugging the calibre of both vessels at their fusion, being the source

of their aneurismal distension. These arteries, and those at the base of the brain were in a state of fatty and atheromatous degeneration.

The right vertebral artery, for a short distance divided in two branches, soon reunited in a single trunk before giving origin to the basilar. The spinal accessory nerve passed through this dicotomic division, and the roots of the glosso-pharyngeal, as well as those of the hypoglossus, were compressed by the aneurismal tumor, figure 1, Plate VIII. This strange disposition accounts for the dysphagia and paralysis of the tongue.

The cortical substance of the brain, anæmic and with a yellow lustrous color, had undergone a general change. The white substance was equally altered, and both corpora striata presented tracts of yellow softening, continuing downward into the central regions of the Pons Varolii. The brain tissue was in a general state of sclerosis, arrived to its highest degree in the superior and inferior marginal convolutions and along the fissure of Sylvius on either side. Under the microscope the nervous elements appeared deficient and replaced by a multiplication of connective cells and fibres.

In the medulla oblongata, the olivary bodies were principally involved by the sclerosis. Ganglionic cells irregular and granular, with their nucleus scarcely distinguishable. The spinal cord, carefully examined in all its length, presented in the cervical and lumbar enlargements, many sections with the whole gray matter reduced to a gelatinous mass of nuclei, granular corpuscles and connective fibres, without nervous cells, and irregularly fused with the surrounding posterior columns. The anterior and lateral columns exhibited plates of amyloid degeneration alternating with regions quite uninjured in their structure. This amyloid degeneration could be distinctly followed up into the gray substance of the Pons and the corpus restiforme of the corresponding side, and even into the peduncles of the cerebellum, although in this latter region the amyloid bodies considerably decreased in amount in the midst of granular corpuscles, connective elements, and sound fibres from the posterior median columns. As to the posterior columns, they were more generally altered at the cervical region, and in many places involving the very superficial fibres of the cord. The spinal ganglia,

and the cervical sympathetic, displayed the same hypergenesis of connective elements, with the cells fatty and infiltrated with pigment. This pigment infiltration and the connective elements were most remarkable on the spinal ganglia connected with the fifth and sixth upper intercostal nerves, animating the cutaneous regions involved by the herpes zoster. The ganglion cells not only exhibited an unusual amount of pigment masking their nucleus, but were besides in a fatty condition, manifested by their brilliant appearance. It is unnecessary to insist upon the hystological changes of these parts, described page 113. Finally, it is worthy of note that, with the above local disorganization of the spinal gray substance, the patient did not complain of anæsthesia in any of the limbs.

I may also remark that the granulation-corpuscles, abundant in this instance, and which I have alike noticed in most of the preceding cases, do not originate always "from cells which are gradually undergoing a process of retrograde fatty metamorphosis preliminary to a complete molecular disintegration," as recently assumed by H. Charlton Bastian, in a most elaborate and interesting paper on a case of Concusion-Lesion with extensive Secondary Degeneration of the Spinal Cord.* I have represented before in figure 2, Plate v, a faithful copy of a specimen, where the fatty molecules grouped around a free connective nucleus, manifest the commencement of a granulation-corpuscle, and this very early stage of the corpuscles I have detected, not only in the instances here alluded to, but in others of degeneration of the brain or spinal cord. I have further met with small granulation-corpuscles, in which, as noticed by Bastian, the original cell wall has been very distinct, particularly after tinting the specimen with carmine. Yet, inasmuch as these smaller corpuscles appeared either contained in, or extravasated from, the adventitious sheath of the capillary vessels, I have looked upon them as lymph-cells running into the prevailing granular metamorphosis previous to their disintegration. Therefore, I am inclined to admit as correct the views held by Bouchard, in his admirable Researches on Secondary Degeneration of the Spinal Cord,† *i. e.*, that such granulation corpuscles are formed by the aggregation of

* Medico-Chirurgical Transactions, vol. L, London, 1867, p. 528.

† Archives Gén. de Méd., Mars, 1866, p. 284.

fat granules resulting from the molecular disintegration of the contents of the nerve-tubes, or from the fatty granular transformation of the myeline globules. Nevertheless, I should not ascribe such a restricted source to the constituent fat granules, for I think that they more abundantly proceed from the retrograde degeneration pervading throughout the anatomical elements and inducing the molecular disintegration of the nervous tissue and exudations therein produced.

CASE XV. *Epilepsy. Hemiplegia with anæsthesia, and sight and hearing impaired on the right side. No trouble with speech. Death. Lesion of the brain, involving the left anterior convolutions, and of the medulla oblongata.*

Elizabeth . . . . . , aged thirty-five, unmarried, born in England, and of intemperate habits, entered the hospital October 8th, 1866. She does not exactly know the time of invasion of the fits, which have existed for many years, but says that three years ago, while at the Alms House, she was locked out doors one night during the winter, and then became paralyzed. The fits are preceded by vertigo, usually coming on every two weeks, and most frequently about the menstrual period. She does not bite the tongue, turns very pale when attacked, and the healthy is the only side seized with spasms, Consciousness retained during the fits; she knows those around her and can speak while convulsed. Headache follows the paroxysm. The skull is rather small: circumference 21 inches; from one to another auditory foramen, passing over the vertex 11½ inches; from the root of the nose to the occipital protuberance 13 inches. Mind impaired. Face and neck covered with minute petechiæ. Speech in no way disturbed excepting as to the distinct articulation of lingual letters, on account of marked paralysis of muscles of the tongue. Sight and hearing defective on right side. Sensibility lost on the right but normal on the left side. Temperature of the limbs low and easily influenced by atmospheric changes. Perspiration deficient. Circulation sluggish. Pulse 89, weak and irregular. Digestive functions not very active; habitual constipation. Urine acid with normal gravity. No irregularity with uterine functions.

I do not think it necessary to enter into the details of treatment: suffice to state that it was wholly unsuccessful, consisting in the

exhibition of half a drachm of bromide of potassium, three times a day, cold shower baths, nutritious diet, and occasional aperients to regulate the bowels. After complaining, in October 20th, that the palsied leg pained her, and that she failed to use it as well as formerly, she completely lost the power of the left limbs, became very crazy, and having had fourteen fits since October 8th, finally died the 30th of December.

Autopsy.— Cadaver rigid and frozen, temperature below 32° Fahrenheit. Pupils equally dilated. White cicatrix at upper and external surface of right thigh. Hair discolored on the right side of the pubis just to the median line, and slightly gray all over the head. Thoracic and abdominal viscera normal, uterus bicornous. Calvarium not thickened or congested. Dura-mater adherent through a limited space to the upper surface of the right hemisphere. Meningeal capillary congestion, more marked in the posterior part of the brain. Small amount of clear cerebro-spinal fluid, pia-mater opalescent, not attached to the cerebral surface. Weight of the encephalon, 48⅜ oz. Brain proper, 43½ oz. Cerebellum, 4 oz. Pons medulla, 1½ oz. Nothing to be noticed in the pineal gland. Cerebral convolutions ill defined. Gray substance, pale, much diminished in width, and fused with the white. Capillary congestion throughout the hemispheres, especially on the right side. Rusty colored patch of an old apoplectic effusion at the posterior part of the left thalamus opticus. The corpus striatum, the medullary substance in the superior marginal convolution, the Insula, as well as the rest of the left cerebral hemisphere, in farther advanced stage of yellow softening than the corresponding parts on the right side. Cerebellum with many red points on section and degenerated. The olivary bodies presented yellow indurations, and both vagal and the right hypoglossal nuclei had a reddish discoloration, due to granular exudation from the distended capillaries. Deep fatty change of the whole sympathetic ganglia in the neck and abdomen.

From all that has been heretofore stated it results that in epilepsy the left as well as both third frontal convolutions may be deranged in their structure without necessarily causing aphasia, which may still be pre-

sent with lesion of both anterior lobes or other regions of the brain. I, of course, could strengthen this assertion with the instances of lesion of the Pons Varolii, or of other cerebral diseases, with loss of speech and no structural change of the third left frontal convolution. But this is a subject to which I purpose to revert at some future time, with a greater number of facts than those I have already studied, in order to arrive at conclusions which might not be judged premature. Concerning the influence of disease of the Pons Varolii on the faculty of speech, Gubler has already referred to it in his valuable memoir on alternate paralysis. Thus he advances: "Utterance will be difficult not from want of memory or of the faculty of language, but because the tongue and other parts of the vocal apparatus, fail to obey the command of will. There will be no incoherence of language, because the patient, aware of his powerlessness to make himself understood, will travel from one to another idea always expecting to succeed in rendering better the last one. Lastly, he will be in a very natural agitation caused by this disease, which, though not depriving him of mind, yet restrains his means of expression. All this could not constitute a true derangement of the mind, it only bears an appearance of it."*

If we now return to the subject of cardiac disease and obliteration of the cerebral blood vessels we find

* Sur les Paralysies Alternes en Général et particulièrement sur l'Hémiplégie Alterne. Paris, 1859. p. 61.

that they accompany each other in five of my cases; with them however, uniliteral spasms were only once particularly observed. The frequency of impediment to the blood current by plugging of the middle cerebral artery has been very notable; yet, the veins have been also obliterated and cardiac disease did not invariably give rise to these phenomena, otherwise induced either by a cachectic state of the blood, or by alcoholismus, which, as already known, contributes so much to coagulation of the blood in the circulatory system. Occlusion was met with twice in the right, and once in the left middle cerebral artery, once in the right vena Galeni, once in the straight sinus, inferior longitudinal sinus and vena Galeni, and once in the vertebral arteries. Atrophy of the cerebral hemisphere and obliteration of the middle cerebral artery have always coexisted, being, as already explained by Kirkes, due to the few anastomoses of the latter artery with the other terminal branches of the carotid.

In another communication,* Dr. Jackson makes reference to cases of epilepsy in which loss of smell and aphamia have attended the epileptic convulsions. One of the patients had valvular disease of the heart, and Dr. Jackson thought, that at least it was probable, that the left middle cerebral artery was plugged. "Considering that the said artery supplies the roots of the olfactory bulb, the corpus striatum and the

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*Epileptic Aphamia with Epileptic Seizures on Right Side. Medical Times and Gazette, August 13, 1864.

cerebral hemisphere, it may be easily understood how the three above symptoms may happen together on account of the community of nutrition of these different regions, or how vascular contraction impairing nutrition might be limited to one of the arterial regions of the brain." The parallel drawn by Dr. Jackson between the symptoms in such cases of epilepsy and those of arterial embolism rests on pure speculative views not sustained by any autopsy. I would not question the correctness of the interpretation given by Dr. Jackson to the above phenomena, nay, I believe that most probably it is the true one in special cases; but I hardly think that the theory stands any broad application, and the remarks elicited concerning aphasia suffice to make us doubt of it. Moreover, loss of smell may as well result from morbid changes in the Schneiderian membrane, capable of being induced by trouble of the sympathetic, without cerebral derangement, and itself essential to the epileptic disease. It may be possible to diagnosticate in unilateral epileptic seizures, connected with cardiac disease and hemiplegia, the existence of embolism in the cerebral arteries; but the accident frequently may and does happen without originating fits; on this account, therefore, the theory of arterial regions in epilepsy is only admissible for restricted forms of the disease, or more correctly, as Dr. Jackson says, of epileptiform convulsions coincident with hemiplegia. I have previously stated that convulsions and delirium, though mediately depending on disturbance of the cerebral circulation,

do not, however, recognize this as their single chief source, for there are modifications in the action of the nervous elements preliminary to the circulatory trouble and having more prominent influence in the causation of the above morbid phenomena.

Case XIV, is a rare instance of hæmatorrachis and aneurism in both vertebral arteries, substantiating the manner in which the dilatation is occasioned by plugging of the vessel, as first explained by Dr. J. W. Ogle. A condition identical with that here represented, may exist in the small capillaries, where minute granules, either carried by the blood current or generated in situ, give rise to their obliteration and dilatation. In many instances, the blood seems to have suffered a coagulation in mass in the contracted portion of the vessel, probably during the period of peripheral circulatory arrest attending the epileptic paroxysm. These aneurisms I have usually detected whenever the brain tissue has displayed a punctiform or capillary congestion. But then, the greater number appeared produced by rupture of the proper tunics and extravasation of blood into the cavity of the adventitious sheath of the vessel, figures 2 and 3, Pl. VI. They exhibited the characters of *dissecting* or *spurious aneurisms*, as first described in apoplexy, by Pestalozzi.* Similar expansions are encountered in the capillaries connected with the minute ecchymoses or

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*Ueber Aneurysmata Spuria der Kleinen Gehirnarterien und ihren Zusammenhang mit Apoplexie. Würzburg, 1849, pp. 7 and 13.

petechiæ in the face and neck of epileptics, indicative of previously existing attacks. In these places the over distended tunics of the vessel are torn asunder, and the blood extravasated in the surrounding cellular tissue; not unfrequently the extravasation is located in the very vascular loop of the papillæ of the skin. Plate x represents the capillaries from one of the laminæ of the cerebellum in the last case of the table of autopsies. Some of the vessels exhibit the aneurismal dilatation, while the majority retain their regular caliber and the curious arrangement, which has been accurately copied with the camera lucida, after staining the blood vessels with carmine. The capillaries are usually in a state of fatty degeneration in the bluish or dull purple stains of the skin, particularly observable in the lower more than in the upper limbs, and with inveterate cases of epilepsy.

The metamorphosis of the cerebral capillaries may be partial or extending over the whole length of the vessel. With the capillaries of the first and second varieties, of Robin, the change commences at the inner coat, but in the largest, or capillaries of the third variety, the degeneration commonly sets in first on the adventitious sheath, figure 1, Plate vi. The muscular fibres, pressed asunder by the dilatation of the vessel, exhibit their interstices filled by heaps of fatty granules, which gradually invade the nuclei and external layer, thickened by the proliferation of connective elements. It is in the more constricted parts of the vessel that the muscular elements last longer. The



Adina [illegible]

BLOODVESSELS OF CEREBELLUM EPILEPSY

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vessel may become completely plugged by granulations, white cells and blood corpuscles, which soon undergo a retrograde change, forming a fatty mass tinged with hæmatic crystals at the site of the above constriction. In the atheromatous degeneration, attended with the same sclerosis of the external coat, the nuclei and fatty elements are mixed with albuminous and calcareous granulations, and crystals of cholesterine which, when accumulated, give a glistening silver like aspect to the atheromatous mass. The calcareous matter may, however, be spread through the whole structure of the vessel as in Case VIII, in which the cerebral capillaries underwent a complete calcification.

The blood from epileptics dying in a fit has a dingy red color, with soft grumous clots, pale blood corpuscles, and the serum tinted with the coloring matter of the corpuscles. Although usually the corpora amylacea, in cases of epilepsy, appear very much increased in those regions of the nervous centres where they normally exist, and that I have also met with the amyloid degeneration of the blood vessels, yet, I have never discovered any corpora amylacea in the blood taken from the living or dead body of epileptics. Since Stratford* published the discovery of numerous amyloid bodies in the blood of an epileptic, Dr. Wm. A. Hammond † has also reported two cases in which

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* Quarterly Journal of Microscopical Science, vol. III, 1855, p. 168.

† The Maryland and Virginia Medical Journal, April, 1861, p. 271.

he observed the same phenomenon in the blood of two female epileptics. In one of the patients the bodies were present in the blood for a period of ten hours (four hours before six hours after a fit) and at no other time. I am not aware of any more instances of the kind, and have never detected any corpora amylacea in the blood of epileptics, whether examined before, during, or after a fit; or taking it from the neck, the petechiæ at the face, or from any other part of the body, including the blood vessels of the brain and medulla oblongata itself. The same negative experience has been obtained by Virchow* who believes — and I concur with him — that such starchy corpuscles, as shown by Rouget, are merely accidentally derived from external sources.

Local paralysis, with contraction or deformity of the limbs, not unfrequently accompanies congenital epilepsy, and, in my belief, acknowledges its source in lesion of the sympathetic. I do not, however, reject the occurrence of such accident upon disease of the brain or spinal cord. I previously pointed out the immediate absence of cadaveric rigidity and earlier putrefaction of the muscles in the palsied limbs of epileptics, adding. that such phenomena were more notorious in those muscles seized with rigidity during life, and I may further assert, that they likewise occur, independently of any previous paralysis, when death supervenes upon a succession of fits. But, aside of

* Cellular Pathology, p. 420.

these interesting peculiarities, there are structural modifications of the muscular elements, which deserve notice. Reference has been made in Case VIII, to the degeneration exhibited by the palsied limbs, and to its analogy with the myo-sclerosic paralysis of Duchenne de Boulogne. Identical lesion I have detected in other instances of epilepsy, the hystological change being usually constituted by a granular disintegration of the muscular fibres, or its gelatinous transparency, with increased number of nuclei in the myolema. A fatty metamorphosis prevails along with either change and the hypergenesis of connective elements. Moreover, the cardinal point in regard to this local trouble of the muscular system is, that it appears in no essential manner related to the spinal cord or brain; for, usually, microscopical examination shows the degeneration in its various stages from the periphery to the ganglia, beyond which it ceases to be definite. For this reason, I have been led to think that, to the sympathetic, and not to the spinal system, ought we to ascribe the principal origin of the muscular lesion in local palsy attending epilepsy, as well as in infantile, reflex, and other kinds of peripheral paralyses.

Finally, I need not remark that the pituitary body, regarded by Wenzel as the seat of epilepsy, has been examined in every case here reviewed, without discovering any particular malformation of the sella turcica, or the diseased conditions of the body, described by Wenzel.

Synoptic Table of the Post-mortem

Sex.		Age.	Cause.	Nature of Disease.	Chest.
	Female.	12	Unknown. Syphilis?	Epilepsy and alternate hemiplegia.	Enlargement of the heart. Deposits in mitral and aortic valves. Pleuritic adhesions on right side; lower lobe of right lung emphysematous.
Male.		54	Intemperance.	Epilepsy and left hemiplegia.	Tuberculosis of lungs.
	Female.	40	Epileptic from childhood, and intemperate. Origin of fits unknown.	Epilepsy with palsy and contraction in right limbs. Convulsions unilateral.	Heart soft and dilated; atheroma of aortic arch and valves. Congestion of lungs; right lower lobe hepatized.
	Female.	38	Unknown.	Epilepsy and dementia. Bed sores over sacrum, spinous processes exposed. Œdema of lower extremities.	Heart and lungs normal; coagulum thick and firm, filling the right cardiac cavities and pulmonary arteries.
	Female.	37	Unknown.	Epilepsy and dementia.	Melanotic and tuberculous pulmonary deposits. Enlargement of the heart. Coagulum in right cavities.
Male.			Syphilis.	Epilepsy with neuralgia and paralysis of lower limbs.	

Examinations of 26 Epileptics.

Abdomen.	Head.	Remarks.
Uterus nulliparous. Orifice of urethra swollen and ulcerated.	Fatty tumor of dura-mater. Aneurism of right middle cerebral artery. Degeneration of the brain, cerebellum, and medulla. Sclerosis of Pons Varolii. Nucleus of right facial injured. Capillaries more distended in the hypoglossal nuclei, and in the path of the right pneumogastric. Sympathetic not examined.	Death after a fit.
	Neomembrane of dura-mater; granulations in the meninges over cerebellum. Middle cerebral arteries and plexus choroid atheromatous. Fatty degeneration of right corpus striatum, optic thalamus and convolutional gray substance. Medulla and cord congested, the first degenerated. Sympathetic not examined.	Death upon a succession of fits.
Liver contracted and granular. Kidneys fatty.	Neomembrane of dura-mater. Cerebral capillary congestion. Old apoplectic effusions in right corpus striatum, cerebral hemispheres and cortical substance. Speckled ecchymoses in the insula and third left frontal convolution. Plugging of the right vena Galeni, with distension and rupture of right vena corporis striati, and extravasation in the ventricles. Yellow softening of the cerebellum and medulla oblongata, preponderant in the vagal nuclei. Bones of skull thickened.	Death in a fit. Never exhibited signs of aphasia. No examination made of the sympathetic.
Liver fatty. Kidneys granular. Suprarenal capsules disorganized into a chocolate color substance. Uterus enlarged. Granular cervicitis. Coagulum in inferior vena cava.	False membrane in the cavity of arachnoid along left fissure of Sylvius. Anterior convolutions flattened. Punctiform cerebral congestion and softening of left cerebral hemisphere and cerebellum. Degeneration of the medulla and sympathetic.	
Adventitious patches over the liver and spleen, this latter indurated. Kidneys and suprarenal capsules diseased. Carcinoma of neck of the uterus and bladder. Pelvic abscess.	Patch in the meninges over the left Sylvian fissure. Softening of the corpus callosum. Pisiform cavities in either centrum ovale. Apoplectic effusions in left corpus striatum, and in convolutional gray substance of left anterior and middle lobes, with deep lesion of the insula and the superior marginal convolution connected with the patch. Anterior branch, and small division near the origin of the left middle cerebral artery, atheromatous and impervious. Medulla degenerated, greater injury in the vagal nuclei. Sclerosis and amyloid degeneration of sympathetic.	No aphasia.
	Gummy tumor of dura-mater. Induration and increased size of the brain. Complete occlusion of longitudinal sinus. Amyloid and fatty degeneration of the brain and medulla oblongata.	Encephalon only examined.

Sex.		Age.	Cause.	Nature of Disease.	Chest.
	Female.	26	Syphilis.	Epilepsy — Ptosis and paralysis of limbs on left side. Aura from fingers of left hand. Roseola and mucous patches in the genitals.	Tuberculous deposits in both lungs. Pleuritic adhesions and gray hepatization in left side.
	Female.		Intemperance.	Epilepsy; right hemiplegia with muscular contraction and aphasia.	
Male.		20?	Congenital.	Epilepsy, idiocy, palsy and contraction of right limbs.	Pulmonary tuberculosis.
	Female.	12	Sequelæ of dysentery.	Epilepsy. Tottering gait. Hyperæsthesia of the skin.	
Male.		3	Teething.	Epilepsy.	Subpleural ecchymoses, with mingled red and gray hepatization, and emphysema of the lungs.
	Female.	22	Fright.	Fits at every menstrual period.	Pleuritic adhesions over both lungs, with tuberculous infiltration. Heart normal but flabby.
	Female.	1	Unknown.	Fits preceded by yawning.	

Abdomen.	Head.	Remarks.
Liver fatty. Uterus enlarged, granular cervicitis. Mucous patches in labia minora.	Serous effusion in the arachnoid. Right middle cerebral artery plugged. Sanguineous extravasation over the middle lobe of the right cerebral hemisphere. Softening of the convolutional gray matter. Patches of yellow softening in the right centrum ovale, surrounded by softened tissue. Granulations in choroid plexus, in the ependyma of fourth ventricle and in pia-mater of the medulla. Degeneration of the medulla, more advanced along the path and nucleus of the left hypoglossus. Roots of right pneumogastric, and fibres of the abducens degenerated. No change in the median and ulnar nerves of left arm. Sclerosis of sympathetic.	
	Brain tissue anæmic. Ventricular hemorrhage. Fatty degeneration of the middle cerebral arteries, and of the choroid plexus. Fatty change of the whole encephalon, more advanced in the anterior cerebral lobes. Greater dilation of the capillaries in the vicinity of the vagal nuclei. Sclerosis of sympathetic.	Died in a fit.
Stricture of the colon.	Skull thick and scanty in diploe. Membranes bloodless. Convolutions flattened. Unilateral atrophy of the brain and of the spinal cord. Calcification of the capillaries in the left centrum ovale; left hemisphere fatty, the right in a state of sclerosis. Atrophy of left anterior pyramid. Amyloid and fatty degeneration of the medulla and spinal cord. Right ganglionic roots slighter than the left. Sclerosis and fatty degeneration of the muscles and nerves of the palsied limbs. Bones in right side smaller than in the left. Pigment infiltration and amyloid change of sympathetic.	
	Cerebral congestion. Cholesteatoma in the centre of left cerebellar hemisphere. Lesion of the medulla and of the gray spinal substance; amyloid degeneration in the floor of fourth ventricle, the peduncles of the cerebellum and the medulla. Origin of the hypoglossus principally damaged.	Head only examined.
	Skull with ossified fontanelles. Membranes congested. Tissue of the brain and cerebellum unhurt. Degeneration of the medulla, pneumogastrics and sympathetic.	Death from double pneumonia.
Uterus enlarged, with superficial abrasion of the neck.	Cerebral membranes bloodless. Brain and cerebellum normal. Medulla oblongata and principally the olivary bodies in granular state of degeneration, equally involving the origins of the vagus and hypoglossus. Cervical sympathetic, and solar and lumbar ganglia, changed in structure.	
Entero-colitis.	Brain and cerebellum normal. Medulla oblongata and sympathetic degenerated; abdominal ganglia with pigment infiltration.	Death from diarrhœa.

Sex.		Age.	Cause.	Nature of Disease.	Chest.
Male.		24	Attempt to commit suicide by strangulation.	Epilepsy, with dysphagia and dyspnœa.	Pulmonary tuberculosis.
	Female.	35	Unknown.	Epilepsy with hemiplegia, anæsthesia and sight and hearing deficient on right side.	
Male.		67	Since youth, origin unknown.	Epilepsy, with paralysis of the tongue and dysphagia.	Heart enlarged, atheromatous degeneration of the aortic valves and arch.
Male.		16	Was too young when he had the first fit, did not know cause of it. Excessive pipe-smoking increased the severity and frequency of fits.	Epileptic, demented and crying after fit.	Phthisis pulmonalis. Heart enlarged, aortic valves thickened.
	Female.	31	Intemperance.	Epileptic, and always delirious after fits.	Heart fatty; mitral and tricuspid valves thickened. Pleuræ adherent to lungs, in last stage of pneumonia.
Male.		53	Syphilis.	Epilepsy. Tertiary symptoms, gummy tumors in muscles of the chest; contractions of the arms.	Heart flabby and enlarged. Lungs tuberculous.
Male.		9	Since birth.	Epileptic. Intellectual faculties deficient, and most idiotic.	Heart hypertrophied. Lungs congested.

Abdomen.	Head.	Remarks.
	Brain and cerebellum normal. Speckled extravasation in the medulla, at the olivary bodies, and at the origin of the vagus and hypoglossus. Gelatinous degeneration of spinal gray matter.	Head only examined.
Uterus bicornous.	Brain and cerebellum, as well as the medulla degenerated. Nuclei of pneumogastries and right hypoglossus involved. Lesion of third left frontal convolution.	No aphasia. Deep alteration of the whole sympathetic ganglia.
	Brain dry and anæmic. Aneurism of both vertebral arteries with rupture and hæmatorrhachis. Arteries at the base of the brain fatty. General sclerosis of the brain and cerebellum, arrived to its maximum in the superior and inferior marginal convolutions on either side. Medulla oblongata with olivary bodies principally affected. Sclerosis and disorganization of the spinal gray substance. Spinal gauglia with pigment infiltration and hypergenesis of connective elements, more abundant in the ganglia of the cutaneous nerves distributed at the base of the breast covered with herpes zoster. Sympathetic, displaying similar sclerosis.	Death occurred suddenly.
Liver fatty, weighing 4 pounds 11 ounces.	Skull congested and thicker than usual. Membranes congested. Apoplectic capillary effusion throughout the brain and cerebellum. Medulla congested and degenerated. Capillary enlargement predominant in regions of vagus. Sclerosis and pigment infiltration of the sympathetic.	Died during the night, without premonition.
Liver and kidneys fatty.	Meninges and brain bloodless. Straight sinus plugged by a clot extending into the inferior longitudinal sinus and vena Galeni. Tissue of the brain and medulla with fatty degeneration. Pneumogastrics and sympathetic very much altered.	Died with pleuro-pneumonia.
Liver enlarged and fatty.	Skull reduced to outer table near the upper posterior angle of right parietal, corresponding to a gummy tumor in dura-mater. Considerable subarachnoid effusion of blood. Brain so much softened that it could not maintain its shape six hours after death. Medulla indurated. Cerebral arteries fatty; choroid plexus of right side disorganized and nearly destroyed.	The microscopical examination of the nervous centres was not made.
Stomach and intestines inflamed. Liver gorged with blood. Mesenteric glands enlarged.	Skull thick and deformed. Dura-mater adherent and congested. Subarachnoid serous effusion. Convolutions flattened. Capillary congestion, with a remarkable number of dissecting aneurisms throughout the brain and cerebellum. Cortical and white substances blended in many places, and displaying a general sclerosis. Medulla oblongata and sympathetic degenerated, with great hyperplasia of connective elements in the solar plexus, and disintegration of the ganglionic cells.	Death from exhaustion upon diarrhœa.

Sex.		Age.	Cause.	Nature of Disease.	Chest.
Male.		43	Intemperance.	Epilepsy and left hemiplegia.	Pericardium filled with serosity, heart enlarged and fatty. Lungs hepatized.
	Female.	6	Since birth.	Epilepsy and idiocy.	Heart and lungs normal.
	Female.	35	Unknown.	Epilepsy and dementia.	Heart small, lungs normal.
	Female.	68	Unknown.	Epilepsy and dementia.	Left lung tuberculous, pleuritic adhesions on the right. Left ventricle very much hypertrophied.
Male.		18	Unknown.	Epilepsy lasting one year.	Hypertrophy of the heart. Phthisis pulmonalis.
	Female.	5	Scarlatina.	Epilepsy, at first nocturnal and afterward repeating at any hour of day or night, preceded by a piercing cry. Fits lasting two and a half years.	

Abdomen.	Head.	Remarks.
Kidneys fatty.	Skull very thin on the parietal regions. Membranes thickened and friable. Convolutions atrophied. Softening of right cerebral hemisphere. Apoplectic effusion of recent date in right corpus striatum. Ventricles distended by opaline serosity. Granulations in choroid plexus. Medulla degenerated; regions of hypoglossi more injured. Sclerosis and fatty change in cervical sympathetic.	Died with œdema of the limbs, and from exhaustion.
Spleen enlarged. Small intestines with glands of Peyer ulcerated.	Skull thick and compact. Membranes thin and adherent, very much congested. Convolutions flattened. Tissue of the brain and cerebellum firm, elastic, and not very vascular. Softening in the centre of left corpus striatum. Cortical substance very thin, in many points hardly one line wide; left olfactory nerve quite rudimentary. Medulla oblongata and sympathetic very much altered.	Died with typhoid fever.
Intestinal mucous membrane soft and congested.	Skull very thin, reduced to a transparent table in the left parietal region. Membranes attached to the convolutions. Gray matter very deficient. White substance stuffed with minute cavities, some of them having a rusty color. Similar condition in cerebellum. Medulla and sympathetic deeply injured. Great varicosity of capillaries in the regions of the vagus.	Died after several fits during the night.
Fatty liver. Spleen enlarged. Intestines contracted.	Skull thick; membranes opaque and thickened. Convolutions depressed. Gray and white substance fused together. Anæmic state of the brain in advanced stage of fatty degeneration, as also the cerebellum and medulla oblongata. Lesions more prominent in the vagal than in the hypoglossal regions. Sclerosis and fatty degeneration of the sympathetic.	Death following coma after a succession of fits.
Intestinal adherences. Kidneys contracted. Gelatinous degeneration of the solar ganglia, also present in the cervical sympathetic.	Skull normal. Membranes congested. Cerebral tissue quite natural in appearance and under the microscope. Partial exudation and granular degeneration in the crura cerebelli and left cerebellar hemisphere. Medulla with capillary disfigured and altered; rank growth of connective elements and corpora amylacea, and greater aneurismal dilatation in the course and origin of hypoglossus.	Death from exhaustion.
	Membranes and brain bloodless. Brain tissue without any impairment of structure. Cerebellum with capillary enlargement and granular metamorphosis in both central ganglia, though not uniformly extended, nor very far advanced. Medulla with the characteristic alteration, and more considerable vascular dilatation in the origin and course of the hypoglossi. Abundance of amyloid corpuscles in the olivary bodies and throughout the rest of the organ.	Death from diphtheria. Could only examine the head.

CHAPTER III.

ANALYSIS OF CASES—AGES—HEREDITARY INFLUENCE.

Three hundred and six cases, which have come under my immediate observation, furnish the subject of this chapter. Of them, one hundred and thirty-four were patients at the Hospital for Epileptics and Paralytics, fifty-one at the Charity Hospital and Dispensary of the House of Mercy, and the remaining one hundred and seventeen, private cases. To Drs. Fred. A. Castle, J. McDonal McClung, and L. B. Edwards, I am indebted for most valuable aid in keeping a careful record and for the management of every case at the Hospital for Epileptics and Paralytics, to which they were the Assistant Physicians. Portion of the materials referring to the patients in this latter hospital, served already for the imperfect official report for the last quarter of 1866, sent to the Board of Commissioners of Public Charities and Correction, Dec. 31, 1866, and the whole notes I am about using were collected to draw up a more elaborate report for 1867, which I could not present to the Board of Commissioners.

The sex of the 306 cases is thus divided:

	MALES.	FEMALES.
Hospital for Epileptics, - - - - -	34	104
Charity Hospital and Dispensary, - -	36	15
Private cases, - - - - - - -	60	57
Total, - - - - - - - -	130	176

There is a cause to be taken into account concerning the greater proportion of females from the Hospital for Epileptics, namely, that they were admitted into the institution from its establishment, whereas, the male department was opened at a later date, and, in addition, temporarily destined in part to cases of paralysis. For this reason, including all but these cases, we find that the proportion between males and females with the other one hundred and sixty-eight is, as four males to three females. If we further consider the mortality of patients in the Hospital for Epileptics, from October 1st, 1866, to September 1st, 1867, we find, that three females died during the quarter ending December 31, 1866, and that from January 1st to September 1st, 1867, there were four deaths respectively among males and females. As regards the rest of the cases here in question, eight deaths occurred in the female side and seven in the male.

It is generally established by French authors, on the evidence of hospital statistics, that females are more prone than males to epilepsy. However, as Sieveking remarks,* this statement is at variance with the results of statistics in general, and with the views of English writers, who are unanimous as to the greater proclivity to epilepsy being on the side of the male sex. If we refer to the last Census of the United States, we find in Table IX of deaths, in the year ending June 1, 1860, according to sex, disease

* Op. cit., p. 106.

and month,* that in a grand total of 501 deaths from epilepsy, 284 were among males, and 217 among females. It is, therefore, evident that more males die epileptic than females, and that in one year, 67 male deaths occurred in excess of female deaths from that cause. There was no age either, as shown by Table v, of the Census, at which the female sex should perish more than the male by epilepsy, notwithstanding the assertion of Portal, who says: "it seems as though after seven years of age more girls than boys die from epilepsy."† It is no less interesting that, according to our Census,‡ "Epilepsy was fatal to 373 in 1850, and to 501 in 1860 (the grand total of deaths in the United States being 39,453). The latter were in the same proportion East and West, but nearly twice as prevalent in the North as in the South. The ratio was in the United States 14, England 53, Scotland 33, Ireland 13, France 28, Frankfort 19, and Brussels 7, in 10,000 of all. In 1850 the ratio was 13 in 10,000 deaths of all known causes."

These data agree with those adduced by Sieveking.§ From a table embracing deaths at each age from epilepsy, in England and Wales, during the seven years, 1848 to 1854, and supplied to Sieveking by Dr.

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* Eighth Census of the United States, under the direction of the Secretary of the Interior. Washington, 1866, p. 134.

† Op. cit., p. 120.

‡ Op. cit., p. 243.

§ Op. cit., p. 107.

Farr; "it would appear that the mortality of males at all ages from epilepsy is 52.26 per cent, of females 47.73 per cent, and that, therefore, 4.53 per cent of male deaths occur from epilepsy in excess of female deaths, from that cause: or to put it in a different way we find that the average male deaths in one year from epilepsy are 961.3, of female 878.1; so that annually in England and Wales 83.2 more males die epileptic than females."

I must not, however, lose sight of the elaborate statistics of the distinguished Girard de Cailleux standing foremost among those furnished by contemporary French authors. The total number of patients treated at the Asylum of Auxerre from 1841 to 1857, amounted to 1506. Of these 148 were epileptics: 91 males and 57 females, and their number of deaths 84, divided: 53 among the males, and 31 among the females. It is, furthermore, established by said statistics that — not solely epilepsy, but insanity in its different kinds is more prevalent among males, lypemania * being the only form in marked degree predominant among the female sex.

To what cause is the difference reducible between the above noted statistics and those supplied by the Parisian asylums is, indeed, difficult to assign. I think with Sieveking, that some undetermined circumstance must exist, which assuredly is corrected

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* Etudes Pratiques sur les Maladies Nerveuses et Mentales. Paris, 1863, pp. 24, 26, 84.

when we draw the grand total of deaths from epilepsy in France — a view corroborated by the interesting tables of Girard de Cailleux. I can not but admit by own experience, that the greater proclivity to epilepsy is displayed by males, whose mortality is likewise predominant. This second statement might seem incompatible with the preceding table of autopsies. However, were we to consider that from October 1st, 1866, to September 1st, 1867, the number of deaths amounted to 4 among 34 males, and to 7 among 104 females at the Hospital for Epileptics, thus giving a rate of 11.76 per cent with the former and of 6.73 with the latter, we might with justice admit that the more we rectify the sources of unreliable calculation the greater grows the proportion of loss on the male side. The fact is confirmed by the following data, kindly furnished to me by Dr. E. Harris, Registrar of Vital Statistics. The total of deaths from epilepsy in the city of New York, during the year 1868, amounts to 53, of which 32 among males, and 21 among females. These numbers in every respect agree with those from the statistics of Sieveking, Girard de Cailleux, and of our last Census. I may briefly assert, upon estimate of personal notes and records of different writers, that the greater mortality among males is not peculiar to epilepsy, it is equally met with nervous diseases generally, death by them prevailing among the males in childhood, adolescence, and old age, though in full manhood it ceases to be as constantly so, the loss being greater on the female side in cases of

paralysis. The following data extracted from the already quoted Table v, of our Eighth Census, materially strengthen this view, and favorably compare with the conclusions arrived at by Tripe, who has shown that: deaths of males prevail upon those of females during the first five years of life, from diseases of the nervous system by as much as 20.5 per cent.*

Diseases.	Under 5 Years.		Under 20 Years.		From 40 to 50 Years.		Over 60 Years.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
Brain,	1687	1317	2300	1839	96	225	186	95
Cephalitis,	3735	2922	4846	3919	101	143	137	63
Chorea,	2	1	15	21	2	3	1	1
Convulsions,	4123	3205	4481	3534	80	75	133	109
Epilepsy,	65	54	111	87	33	21	22	19
Hydrocephalus,	1631	1302	1805	1472	15	5	12	13
Neuralgia,	60	58	121	136	47	58	85	110
Paralysis,	65	62	130	124	172	188	1450	1543
Tetanus,	583	456	774	533	48	15	31	19

I will now exhibit in a synoptic table the principal phenomena with my three hundred and six cases, after remarking that in every instance of paralysis, this symptom supervened consecutive to the epileptic paroxysms, occurrence of paralysis with epileptiform convulsions being accordingly excluded from this table.

* British and Foreign Med. Chir. Review. April, 1857, p. 460.

Synoptic Table of

No.	Age.	Nativity.	Occupation.	State.	Nature of the Attacks.	Complications.
1	18	New York....	.....	S.	Spasms on right limbs. Petit mal.....	Intelligence impaired; limbs cold.
2	18	New York....	.....	S.	Petit mal and spasms	Intellect deficient; limbs weak and cold.....
3	16	New York....	....	S.	General spasms.	Right testicle not in scrotum. Stammering; limbs very cold; intellect weak.....
4	20	N. Carolina...	.....	S.	General spasms.	Limbs cold; intellect deficient ....
5	16	New York....	.....	S.	Petit mal and spasms	Hemiplegia left side; limbs cold ..
6	11	New York....	.....	S.	General spasms.	Eczema in arms and chest; limbs cold..... .....
7	13	New York....	.....	S.	General spasms.	Imbecile; limbs cold and purple ..
8	15	Canada	.....	S.	Absence. No spasms	Memory deficient; circulation sluggish..... .....
9	5	New York....	.....	S.	General spasms.	Intelligence low; extremities cold.
10	18	New York....	.....	S.	Petit mal and spasms	Strabismus; right leg paralyzed; limbs cold.....
11	7	New York....	.....	S.	General spasms.	Intelligence poor; limbs cold .....
12	16	Scotland.....	.....	S.	General spasms.	Aortic regurgitant murmur.....
13	17	New York....	.....	S.	General spasms.	Demented; limbs cold .....
14	30	Ireland	Laborer	S.	Petit mal and spasms	Varices in the legs; limbs cold....
15	38	Ireland	Laborer	S.	General spasms.	Maniacal excitement after fits .....
16	46	New York...	Stone cutter..	S.	General spasms.	Hallucination of sight before fits ..
17	22	Ireland	Laborer	M.	General spasms.	Phthisis; limbs cold . .....
18	26	England.....	Salesman....	S.	General spasms.	Chills precede the fits; limbs very cold.....
19	24	Connecticut ..	.....	S.	General spasms.	Maniacal excitement; insanity....
20	16	New York....	....	S.	General spasms.	Demented; limbs cold ....
21	7	New York....	.....	S.	Petit mal and spasms	Head very small. Intelligence low; limbs cold..... .. ..
22	9	New York....	....	S.	General spasms.	Fits attended by vomiting; feet cold.....
23	4	New York....	....	S.	General spasms.	Right limbs paralyzed six months after first fit; limbs cold .....
24	5	Connecticut ..	.....	S.	General spasms.	Pain at the stomach before fits;
25	10	New Jersey ..	.....	S.	Petit mal and spasms, ...	limbs cold..... Nystagmus and abscence; limbs
26	5	New York....	.....	S.	Spasms in left limbs.....	numb and cold ..... Left hemiplegia and contraction in the leg .....
27	6	New York....	.....	S.	Petit mal and spasms	Contraction in left arm; slight facial paralysis in right side .....
28	30	New York....	Clerk.....	M.	General spasms.	Fits became frequent at the age of 25; limbs cold.....
29	10	New York...	.....	S.	General spasms.	Aura from stomach; limbs cold ...
30	10	New York....	.....	S.	Petit mal and spasms	Limbs very cold and weak .....
31	15	New York....	.....	S.	General spasms.	Vomiting after fits; limbs cold....
32	16	New York....	.....	S.	General spasms.	Palpitation of the heart; limbs cold

Male Epileptics.

Cause.	Age of Invasion.	Hereditary Influence.	State of Pulse.	Remarks.
.....	Birth ...	Mother epileptic.....	65, small....	Addicted to onanism.
.....	.....	Mother epileptic, father phthsie	84, intermittent and soft.....	"Born with fits."
.....	Birth ...	Brothers of father and mother epileptic....	78, irregular, feeble.	
.....	Infancy.	Father paralytic, brother epileptic.....	70, feeble ...	Bites the tongue during fit.
Dentition.....	Infancy.	Mother intemperate ..	72, weak.	
.....	Infancy.	Father paralyzed	89, feeble.	
.....	Infancy.	Mother maniae	76, irregular	Bites the tongue during fit.
Dentition.....	Infancy.	Parents consumptive .	104, weak...	Attacks preceded by perspiration in head and arms.
Dentition.....	Infancy.	Mother intemperate ..	96, weak.	
Fever	Infancy.	.....	87, soft, indistinct.	
.....	Infancy.	Father dypsomaniae..	88, feeble ...	Bites the tongue during fit.
.....	Infancy.	.....	88, irregular.	
.....	Infancy.	Parents intemperate..	92, weak.	
.....	Infancy.	Father intemperate...	80, feeble ...	Fits in infancy, reappearing at the age of 23, once every year until 26, when they became frequent.
.....	Infancy.	.....	88, feeble.	
.....	Infancy.	.....	68, feeble ...	Had petit mal the first years.
Intermittent fever.	Infancy.	.....	98, small.	
Fever	Infancy.	.....	85, irregular.	
.....	Infancy.	Mother's brother epileptic.....	100, feeble.	
.....	Infancy.	Father paralytic and intemperate, brother idiotic.....	86, soft.	
.....	1 year ..	.....	96, weak.	
.....	1 year ..	.....	93, weak.	
.....	2 years .	.....	98, weak.	
....	2 years .	.....	87, weak.	
.....	2 years .	.....	92, soft.	
Diarrhœa.....	2 years .	.....	89, intermittent and feeble.	
Fever	3 years .	....	84, feeble.	
.....	3 years .	.....	78, weak....	Bites the tongue during fit.
Indigestion	3 years .	.....	88, soft.	
Dysentery.....	4 years .	.....	96, small.	
Indigestion	5 years .	.....	76, weak.	
Scarlatina.....	5 years .	.....	88, feeble ...	Bites the tongue during fit.

No.	Age.	Nativity.	Occupation.	State.	Nature of the Attacks.	Complications.
33	16	Rhode Island.	.....	S.	Spasms on left side	Contraction and atrophy of left arm; limbs numb and cold .....
34	28	Germany	.....	S.	General spasms.	Glottic spasms before fits; phthisis
35	21	Rhode Island.	Clerk.....	S.	Rolling spasms.	Strabismus; facial palsy; anæsthesia; diminished temperature on left side; polinria ....
36	9	New York....	.....	S.	Absence. No spasms	Limbs cold; circulation feeble; memory poor.....
37	22	New York....	.....	S.	General spasms.	Deafness on right side; limbs weak and cold .....
38	18	New York....	... .	S.	General spasms.	Limbs cold; memory dull .....
39	12	New York....	.....	S.	Petit mal and spasms	Intellect very low; limbs cold.....
40	15	New York....	.....	S.	Petit mal.	Idiotic; limbs weak and cold.....
41	16	Connecticut ..	....	S.	Petit mal.	Intelligence deficient; circulation imperfect . .. .
42	13	New York....	.....	S.	Petit mal.	Hemiplegia on left side; tingling of the limbs.....
43	16	New York....	.....	S.	Noct. spasms...	Paralysis of right limbs, cold and numb ....
44	20	Ireland	.....	S.	General spasms.	Sight impaired in right eye; limbs cold and numb .....
45	15	New York....	.....	S.	Noct. spasms ...	Circulation imperfect; limbs cold.
46	26	Ireland	Driver.....	S.	General spasms.	Paraplegia after fits; limbs cold...
47	33	New York ...	.....	S.	General spasms.	Maniacal excitement after fits .....
48	30	New York ...	Clerk	S.	General spasms.	Memory lost; limbs cold..... .....
49	20	New York	.....	S.	General spasms.	Hemiplegia on left side; limbs cold.....
50	56	Ireland	Tailor	W.	General spasms.	Occurred in childhood and brought back by intemperance.....
51	31	New York....	.....	S.	General spasms.	Systolic murmur at the base of the heart; cannot articulate distinctly .....
52	16	New York	Druggist	S.	General spasms.	Speech impaired after fits .....
53	39	England	Laborer	S.	General spasms.	Mania after fit; limbs powerless and cold .....
54	21	Island of Cuba	....	S.	General spasms.	Maniacal excitement after fits; Limbs cold .....
55	26	New York....	Clerk	S.	General spasms.	Vertigo and palpitation of the heart ....
56	18	New York ...	.....	S.	Spasms on right limbs.... .. .	Hemiplegia on left side after fits...
57	17	New York....	Ship boy ...	S.	General spasms.	Tingling in the hands before fits ..
58	16	New York....	.....	S.	General spasms	Limbs cold... .....
59	18	New York....	Clerk	S.	General spasms.	Vertigo; cold extremities,.....
60	21	California....	Agent	S.	General spasms.	Tinnitus aurium before fits .....
61	18	New York....	Clerk	S.	Spasms on right limbs.	Paralysis of right arm and eyelid .
62	19	New York....	.....	S.	General spasms	Phthisis; extremities cold.....
63	26	New York....	.....	S.	Spasms in limbs not palsied ...	Hemiplegia left side; paralysis of palate..... .....
64	23	New York....	.....	S.	Noct. spasms...	Circulation imperfect in the limbs.
65	28	England	... .	S.	General spasms.	Limbs cold..... ....
66	23	Ireland	Laborer.	S.	Petit mal; no spasms	Intellect impaired; limbs cold....
67	23	France	Laborer.....	S.	General spasms.	Feet cold . .....
68	27	Holland	Waiter	S.	General spasms.	Extremities numb and cold.....
69	21	New York....	Oysterman....	S.	General spasms.	Legs numb and cold .....
70	23	New York....	Painter.....	S.	Petit mal.	Profuse perspiration and exhaustion after fits.....
71	30	New York....	Lawyer	S.	General spasms.	Foul breath for a day before fit....
72	23	Connecticut...	Farmer	S.	General spasms.	Speech thick after fit; limbs cold..
73	22	New York....	.....	S.	General spasms.	Numbness and coldness in the feet

Cause.	Age of Invasion.	Hereditary.	State of Pulse.	Remarks.
Scarlatina	6 years.		79, small.	
	6 years.	Mother epileptic, died in fit	74, small.	
Fall and wound of scalp	6 years.		108 to 120	Pulse firm, not uniform.
	7 years.	Maternal grandmother idiotic, sister epileptic	95, weak.	
Scarlatina	9 years.		76, feeble	Bites the tongue during fit.
Blow on the head	9 years.		88, small	Bites the tongue during fit.
	10 years		89, small	Bites the tongue during fit.
	10 years		78, weak.	
	10 years		90, feeble.	
Onanism	11 years		96, small.	
	11 years		93, feeble.	
Rheumatism	11 years		64, weak	No sign of cardiac disease.
	12 years		76, feeble.	
Diarrhœa	12 years	Sister epileptic	78, feeble.	
	12 years		64, weak.	
Fever	12 years		68, weak	Bites the tongue during fit.
Fever	12 years		79, small.	
	12 years	Sister and cousin epileptic; none of his parents	70, weak	Bites the tongue during fit.
	13 years	Father epileptic	108, soft.	
	14 years	Father's brother epileptic	86, weak.	
	14 years	Father epileptic; died in fit	74, small.	
	14 years		86, small.	
Onanism	14 years		70, small	Bites the tongue during fit.
Fever	15 years		92, feeble	Bites the tongue during fit.
Blow on the head	15 years	Father phthisic; cousin paralytic	76, firm.	
Excitement	15 years		89, feeble.	
Onanism	16 years		86, feeble	Fits usually occur upon vomiting.
Intemperance	17 years		95, irregular.	
Syphilis	17 years		72, irregular.	
Attempted strangulation	17 years		76, feeble.	
	18 years		85, weak and small.	
Mental over-exertion	18 years		70, weak.	
	18 years		96, feeble.	
	18 years		86, small.	
Anger	18 years		80, natural.	
Fall	19 years		92, weak.	
Fatigue and abuse of tobacco	19 years		85, feeble	Bites the tongue during fit.
Lead poisoning (?).	19 years	Father's sister epileptic	72, feeble	Has never had the painter's colic, nor has any discoloration of the margin of the gums.
	20 years		65, feeble.	
Fall from a cart	20 years		78, irregular	Bites the tongue during fit.
Overwork	20 years		68, weak.	

No.	Age.	Nativity.	Occupation.	State.	Nature of the Attacks.	Complications.
74	26	New York....	.....	S.	General spasms.	Weakness and numbness of legs: cystitis.....
75	25	New York....	Reporter	S.	General spasms.	Feet numb and cold... .....
76	29	New York ...	Tinsmith	S.	Petit mal. and spasms	Numbness in lower limbs; always cold.....
77	30	Germany	Baker	M.	General spasms.	Inguinal hernia; extremities cold.
78	44	Ireland	Sailor	M.	General spasms.	Syphilitic ulcer in right leg.....
79	28	New York....	Clerk	S.	General spasms.	Preceded by cramps in right leg...
80	28	New York....	Photographer.	M.	General spasms.	Eczema in the hands; Inguinal hernia; limbs numb and cold....
81	28	France	Merchant.....	S.	Spasms on left side.....	Tertiary accidents; phosis.....
82	82	New York....	Lawyer.....	M.	General spasms.	Slight strabismus; limbs cold.....
83	27	Scotland.....	Machinist	S.	Noct. spasms ...	Secondary syphilis ....
84	28	New Jersey ..	Carman.....	S.	Petit mal.	Hypochondria; Phthisis.....
85	29	New York....	Mason.....	S.	General spasms.	Speech lost after fits; limbs cold..
86	30	New York....	.....	S.	Spasms on left side	Right testicle not in scrotum; Right hydrocele; limbs cold.....
87	30	Ireland	Waiter	S.	General spasms.	Exostoses in tibiæ and gummata..
88	31	New York....	Carpenter	M.	General spasms.	Delerious after fits .. ..
89	31	Louisiana	Merchant.....	S.	General spasms.	Phthisis; extremities weak and cold..... .....
90	30	New York....	Merchant.....	S.	General spasms.	Phthisis; tremor; limbs cold .....
91	34	England	Agent	M.	General spasms.	Numbness and cramps in lower limbs ....
92	33	New York....	Broker	S.	Noct. spasms ...	Numbness with tingling in hands and feet..... ....
93	36	Ireland	Plumber.....	M.	General spasms.	Memory dull; headache; feet cold.
94	32	Ireland	Laborer	S.	General spasms.	Numbness and cold in the feet ....
95	36	New York....	Salesman.....	S.	General spasms.	Aphasia after fits; limbs cold....
96	34	New York....	Clerk	S.	General spasms.	Legs weak and cold.....
97	34	Ireland	Laborer	S.	General spasms.	Wild and cries before fits.....
98	33	Ireland	Laborer	M.	General spasms.	Spasms begin in right hand .....
99	35	New York....	Shoemaker ...	M.	General spasms.	Tremor and general numbness ....
100	35	Ireland	Mason.....	S.	General spasms.	Fits followed by violent delirium..
101	35	New Jersey...	Peddler.....	S.	General spasms.	Phthisis; limbs cold .....
102	35	New York....	Merchant.....	M.	General spasms.	Tubercles in right lung; limbs cold
103	36	Scotland.....	.....	S.	General spasms.	Numbness of lower limbs .....
104	37	New York. ..	Lawyer.....	M.	General spasms.	Congenital ptosis in right eye; limbs cold.....
105	36	Ireland	Laborer	S.	General spasms.	Melancholy after fits ....
106	37	Ireland	Shoemaker ...	S.	General spasms	Extremities numb and cold.....
107	40	England	Brass finisher.	M.	General spasms.	Chancroid.....
108	36	England	Sailor.....	S.	Noct. spasms ..	Feet numb and cold .....
109	37	England	Plumber.....	S.	General spasms.	Had petit mal in the beginning....
110	42	New York....	Driver.....	S.	General spasms.	Tremor and mania after fits .....
111	40	Germany	Shoemaker ...	M.	General spasms.	Lower limbs weak and cold.....
112	40	Ireland	Laborer	M.	Spasms in upper limb.....	Limbs cold and numb.....
113	40	Ireland	Laborer	M.	Spasms on left limb	Contraction on left limb one year after fits; limbs cold.... .....
114	42	New York....	Broker	W.	General spasms.	Maniacial excitement after fits ....
115	42	Ireland	Laborer	M.	General spasms.	Syphilides; ptosis in left side.....
116	42	Pennsylvania.	Farmer	S.	General spasms.	Blind; loss of smell offensive perspiration after fits; optic nerve atrophied . ...
117	46	Ireland	Painter	S.	General spasms.	Has had delirium tremens after being seized with fits .....
118	48	Ireland	Laborer	M.	General spasms.	Talkative after fits; limbs numb and cold ....

Cause.	Age of Invasion.	Hereditary.	State of Pulse.	Remarks.
Venereal excess...	20 years	.....	97, weak.	
Fatigue and intemperance	22 years	.....	86, feeble ...	Bites the tongue during fit.
Venereal excess...	24 years	Mother died imbecile.	97, feeble.	
Yellow fever	24 years	....	80, soft	Bites the tongue during fit.
Poisoning.....	24 years	.....	65, feeble...	"The natives in India gave him a drug to have fits and he exempted from service."
Typhoid fever.....	24 years	... .	70, feeble ...	
.....	24 years	.....	75, feeble ...	Confusion of mind.
Syphilis.....	25 years	....	68, feeble.	
Overwork.....	25 years	.....	70, weak.	
Syphilis.....	25 years	....	76, small.	
Insolation.....	26 years	.....	70, small.	
Conenssion npou a fall.....	27 years	.....	65, weak.	
Mental disturbance	28 years	.....	88, feeble.	
Syphilis.....	28 years	.....	74, feeble.	
Intemperance	29 years	.....	68, weak.	
Fatigue	29 years	.....	104, small, irregular.	
Intemperance	29 years	.....	69, small.	
Insolation.....	30 years	.....	64, small.	
Anxiety.....	30 years	Brother insane.....	94, weak.	
... .	30 years	....	68, small....	Fits at first nocturnal.
Insolatiou..	30 years	.....	70, weak.	
Fever	30 years	.....	85, feeble.	
Typhoid fever.....	30 years	.....	76, feeble.	
Intemperanee	30 years	.....	92, intermittent.	
.....	30 years	.....	72, small....	Bites the tongue during fit.
"Excessive chewing & drinking"	32 years	.....	60, small	
Insolation.	32 years	.....	68, weak.	
Fatigue and intemperance	32 years	.....	72, weak.	
Intemperance	33 years	.....	100, irregular weak.	
Blow on the back .	33 years	....	69, soft.	
Mental overwork..	34 years	Father's family consumptive..... ...	68, small ...	Most of the attacks at night, hites the tongue during them.
Grief.....	34 years	.....	80, firm.	
Intemperanee	34 years	.....	84, small.	
Intemperanee	34 years	.....	73, small ...	Aura starting from the arms.
Severe punishment	34 years	.....	64, weak.	
Intemperance	35 years	.....	70, weak.	
Intemperanee	36 years	.....	60, small....	Bites the tongue dnring fit.
Grief.....	38 years	.....	64, feeble.	
Intemperance	38 years	.....	80, soft.	
Over work and intemperanee	38 years	.....	60, small.	
Mental disturbauce	39 years	.....	97, quick & small.	
Syphilis.....	39 years	....	70, small....	Bites the tongue during fit.
Fall on the head ..	39 years	.....	98, small....	Sight was gradually lost.
Intemperance	40 years	.....	72, small.	
Blow on the head.	46 years	.....	92, soft.	

No.	Age.	Nativity.	Occupation.	State.	Nature of the Attacks	Complications.
119	49	New York....	Book keeper..	M.	Noct. spasms ..	Hand and feet cold and numb .....
120	50	Ireland	Laborer	M.	General spasms.	Legs weak; numb and cold .....
121	56	Ireland	Laborer	W.	General spasms.	Weakness in left limbs: extremities cold.....
122	50	Ireland	Cartman.....	M.	General spasms.	Hemiplegia left side; limbs cold ..
123	54	Iceland	Laborer	M.	General spasms.	Speech thick; left pupil larger than right; mitral regurgitant murmur.... .....
124	59	Germany	Tailor	M.	General spasms.	Limbs numb and cold.....
125	60	Ireland	Laborer	S.	General spasms.	Numbness and cold in the limbs ..
126	61	New Jersey ..	Merchant.....	M.	General spasms.	Systolic murmur at base of heart..
127	65	Ireland	Laborer	M.	General spasms.	Right limbs slightly paralyzed after fits... ....
128	70	New York....	.....	S.	General spasms.	Paraplegia; numbness and cold in the legs .....
129	70	Ireland	Laborer	M.	General spasms.	Delirious after fits .....
130	33	New York....	Driver.....	S.	Petit mal and spasms.	Dementia; limbs cold.....

Cause.	Age of Invasion.	Hereditary Influence.	State of Pulse.	Remarks.
Intemperance	46 years	.....	58, slow and small.	
Fatigue after march in the army	46 years	.....	86, soft, quick.	
Intemperance	48 years	.....	70, weak and small.	
Intemperance	48 years	....	66, weak.	
Rheumatism	50 years	.....	92, small.	
Hard work and intemperance	54 years	.....	70, small.	
Intemperance	57 years	.....	62, small.	
Rheumatism	58 years	.....	60, slow and small.	
Cerebral concussion upon a fall..	63 years	.....	58, small.	
Intemperance	64 years	.....	62,indistinct.	
Intemperance	66 years	.....	68, soft.	
Intemperance	Unkn'n.	.....	70 soft.	

Synoptic Table of

No.	Age.	Nativity.	Occupation.	State.	Nature of the Attacks.	Complications.
1	30	Ireland.....	Seamstress...	S.	Petit mal and spasms.....	Irregular action of the heart; intellect poor; limbs cold.....
2	40	Pennsylvania.	Dressmaker..	W.	Petit mal and spasms.....	Limbs very cold and purple.....
3	25	New Jersey..	.....	S.	Noct. spasms...	Stammers the day after fits; limbs cold.....
4	?	New York ...	.....	S.	General spasms.	Dementia; low temperature of the body.....
5	5	New York ...	.....	S.	General spasms.	Nearly idiotic; limbs cold.....
6	4	New York ...	.....	S.	General spasms.	Head small; mind undeveloped; limbs cold and weak.....
7	32	England	.....	S.	Petit mal and spasms.....	Action of heart irregular; no murmurs.....
8	20	New York ...	.....	S.	Petit mal and spasms.....	Dysmenorrhœa; granular cervicitis
9	10	New York ...	.....	S.	Petit mal and spasms.....	Imbecile; extremities very cold..
10	27	New York ...	.....	S.	Petit mal and spasms.....	Night blindness; Fits preceded by pain in right side.....
11	9	Vermont	.....	S.	Petit mal and spasms.....	Intellect deficient; limbs cold.....
12	6	New York ...	.....	S.	Petit mal and spasms.....	Slight strabismus; limbs cold.....
13	18	New York ...	.....	S.	General spasms.	Mental faculties low; prolapsus recti; limbs very cold.....
14	12	Wisconsin....	.....	S.	General spasms.	Sweating and increased temperature of right limbs.....
15	5	New York ...	.....	S.	Petit mal and spasms.....	Herpetic eruption in the limbs which are cold.....
16	21	New York ...	.....	S.	Spasms on right limbs.....	Left limbs paralyzed and cold.....
17	25	New York ...	Sick nurse....	S.	General spasms.	Phthisis; varices in the legs; limbs cold; systolic direct murmur at base; mind affected.....
18	38	Ireland.....	Domestic ...	M.	General spasms.	Aortic direct murmur propagated above base; limbs cold; memory dull.....
19	20	Pennsylvania.	.....	S.	General spasms.	Converging strabismus; limbs cold
20	21	New York ...	.....	M.	Spasms only in legs.....	Paraplegia and profuse prespiration after fits; limbs cold .....
21	20	New York ...	.....	M.	Spasms in left limbs.....	Hemiplegia in right side; no trouble with speech; incontinence of urine; limbs cold.....
22	9	New York ...	.....	S.	General spasms.	Intellect low; extremities cold and purple.....
23	15	New York ...	.....	S.	Petit mal and spasms.....	Irregular menstruation; feet cold
24	18	Connecticut ..	.....	S.	Noct. spasms...	Occasional dimness of sight; limbs cold.....
25	17	New York ...	.....	S.	Noct. spasms...	Insanity; cold limbs.....
26	9	Connecticut ..	.....	S.	General spasms.	Slight hemiplegia left side; limbs cold.....
27	30	New York ...	.....	S.	General spasms.	Hearing lost; was insane when 5 years old; limbs cold.....
28	12	New York ...	.....	S.	Spasms in right limbs.....	Hemiplegia in right side after fits; no aphasia; limbs cold.....
29	10	New York ...	.....	S.	Unconscious with't spasms	Facial neuralgia; limbs cold.....
30	8	New York ...	.....	S.	General spasms.	Hands and feet always cold.....

Female Epileptics.

Cause.	Age of Invasion.	Hereditary Influence.	State of Pulse.	Remarks.
.....	Birth ...	Mother intemperate ..	102, intermittent...	Bites the tongue; had part of it amputated.
.....	Birth ...	.....	92, irregular	Spasms only during catamenia; last child died after birth with fits.
.....	Birth ...	.....	68, small....	Bites the tongue during fit.
.....	Birth ...	.....	86, weak....	Bites the tongue during fit.
.....	Birth ...	Mother had severe attacks of agne when pregnant	106, irregular	Bites the tongue during fit.
.....	Birth ...	Father intemperate...	95, feeble.	
.....	Birth ...	.....	100, irregn'r.	
.....	Infancy.	.....	86, feeble ...	Bites the tongue during fit.
.....	Infancy.	Mother epileptic and phthisic; father intemperate	88, irregular weak.	Bites the tongue during fit.
Dentition	Infancy.	Mother phthisic	80, feeble...	Had petit mal until 20 years old.
Dentition	Infancy.	Father died paralytic .	92, irregular.	
Dentition	Infancy.	.....	98, irregular.	
Dentition	Infancy.	Parents nervous.....	97, feeble.	
Dysentery.....	Infancy.	Brother of maternal grandmother insane	96, feeble.	
Dentition	Infancy.	....	94. feeble.	
Dentition	Infancy.	.....	90, small....	Tickling in fingers of left hand before fits.
.....	Infancy.	Parents consumptive.	86, weak....	Bites the tongue during fit.
.....	Infancy.	Mother very intemperate	96, weak....	Two of her children died in fits.
.....	Infancy.	Father died with cerebral softening.....	75, weak....	Bites the tongue during fit.
Dentition	2 years.	.....	90, feeble.	
Fright at the sight of an epileptic..	3 years.	.....	84, weak....	Spasms and palsy since birth of first child at 18; to that time petit mal.
Indigestion	3 years.	Mother's brother and cousin epileptic ...	92, weak.	
Fright by house catching fire.....	4 years.	....	80, weak.	
Blow on the head..	4 years.	.....	92, soft.	
.....	4 years.	Parents phthisic.....	86, irregular	Bites the tongue during fit.
Diarrhœa.....	4 years.	....	94, weak, intermittent	
Scarlatina	5 years.	.....	72, small....	Petit mal until 18 years of age, none thereafter.
.....	6 years.	Mother insane.....	98, weak....	Bites the tongue during fit.
Convalescence from pneumonia.....	6 years.	.....	86, weak....	Bites the tongue during fit.
Fright	6 years.	.....	90, irregular	Bites the tongue during fit.

No.	Age.	Nativity.	Occupation.	State.	Nature of the Attacks.	Complications.
31	23	Ireland	Domestic	S.	General spasms.	Irregular menstruation ; feet cold.
32	16	Ireland	.....	S.	Petit mal and spasms	Phthisis ; limbs cold.....
33	13	New York ...	....	S.	General spasms.	Dementia ; action of the heart irregular ; limbs cold.....
34	10	New York ...	.....	S.	General spasms.	Speechless for two or three hours after fit ; limbs very cold .....
35	14	New York ...	....	S.	General spasms.	Mind impaired ; limbs cold and purple .....
36	12	New York ...	.....	S.	General spasms.	Dementia ; phthisis ; limbs cold ..
37	14	(?)	.....	S.	General spasms.	Phthisis ; limbs cold.....
38	22	New York ...	Seamstress ...	S.	General spasms.	Hysteric ; limbs cold.....
39	11	Rhode Island.	.....	S.	Spasms in upper limbs	Maniacal excitement after fit.....
40	12	New York ...	.....	S.	Petit mal and spasms	Spinal curvature (neck) ; hands cold.....
41	16	New York ...	.....	S.	General spasms.	Left hemiplegia ; idiotic ; systolic murmur at base .....
42	10	New York ...	.....	S.	General spasms.	Fits coming on after chorea ; weakness of lower limbs .....
43	21	England	Domestic.....	S.	Noct. spasms...	Mind impaired ; direct systolic murmur at base ; limbs cold ....
44	18	Ireland	Domestic.....	S.	General spasms.	Dementia ; amenorrhœa ; heart enlarged ; systolic aortic murmur ; limbs cold.....
45	15	New Jersey ..	.....	S.	No unconsciousness ; spasms in right limbs	Atrophy of right arm ; aura from the fingers ; limbs cold.....
46	18	New York ...	Domestic	W.	Noct. spasms...	Amenorrhœa ; chronic cervicitis ; limbs cold .....
47	33	Scotland	.....	S.	Spasms in left limbs	Phthisis ; vertigo and cry before fit ; limbs cold .....
48	18	New York ...	Domestic	S.	Noct. spasms, petit mal in day time	Dysmenorrhœa ; limbs cold .....
49	25	New York ...	.....	S.	General spasms.	Hemiplegia and contraction in right limbs ; phthisis ; extremities cold
50	23	New York ...	Domestic	S.	Spasms in left limbs	Dementia ; emphysema pulmonalis
51	14	New York ...	.....	S.	Noct. spasms...	Vertigo and petit mal in day time ; limbs cold.....
52	20	Ireland	Domestic	S.	Petit mal and spasms.....	Externa strabismus on left side ; systolic murmur most intense at base ; limbs cold .....
53	21	New York ...	.....	S.	General spasms.	Vaginitis ; limbs cold.....
54	15	New York ...	.....	S.	Noct. spasms...	Petit mal in day time ; leucorrhœa ; limbs cold.....
55	18	New York ...	Domestic	S.	General spasms.	Irregular menstruation ; extremities cold .....
56	18	New York ...	.....	S.	General spasms.	Temporary palsy in left limbs ; mitral regurgitant murmur ; limbs cold.....
57	17	New York ...	.....	S.	General spasms.	Dysmenorrhœa ; globus hystericus ; limbs cold.....
58	20	New York ...	.....	S.	Petit mal.....	Speech impeded after fits.....
59	23	New York ...	Seamstress ..	M.	General spasms.	Dysmenorrhœa ; fits before menses ; limbs cold.....
60	23	Ireland	.....	W.	General spasms.	Limbs cold and purple .....
61	17	New York ...	.....	S.	General spasms.	Dysmenorrhœa ; limbs cold .....
62	25	New York ...	.....	S.	General spasms.	Hemorrhoids ; limbs weak and cold

Cause.	Age of Invasion.	Hereditary Influence.	State of Pulse.	Remarks.
Overwork	7 years.	Cousin of father epileptic.....	80, weak. intermittent.	
.....	7 years.	Mother phthisic; father with cardiac disease	115, irregular	Bites the tongue during fit.
.....	7 years.	Father's brother epileptic.....	97, irregular.	
Fright	7 years.	Father paralytic	95, weak....	Bites the tongue during fit.
.....	8 years.	Relatives of mother epileptic.....	79, irregular.	
Fright	9 years.	Mother died with palsy	87, soft.....	Bites the tongue during fit.
Ill treatment	10 years	Mother epileptic.....	84, weak.	
.....	10 years	Brother and mother died apopletic	70, feeble.	
Bite of a dog	10 years	.....	72, irregular.	
Disease of the spine	10 years	.....	97, irregular.	
.....	10 years	Brother idiotic; parents first cousins ..	80, weak.	
Fall on the spine..	10 years	....	89, feeble.	
.....	11 years	Mother epileptic; father phthisic	84, firm.	
"With flood of first meustruation"..	11 years	.....	90, intermittent and indistinct.	
Fall on the arm ...	11 years	.....	76.	Pulse much weaker in atrophied arm.
.....	12 years	Father died with cardaic disease	88, small....	Bites the tongue during fit.
Suppressed menstruation.....	12 years	.....	87, weak....	Bites the tongue during fit.
Establishment of meustruatiou ...	12 years	Father died with cardiac disease	85, intermitteut, full..	Fits occur only during catamenia while asleep.
Excessive punishment..... ...	12 years	Father phthisic.....	87, weak....	Fits preceded by pain in the tongue.
Suppressed menstruation.....	13 years	.....	95, feeble.	
.....	13 years	.....	72, weak....	Bites the tongue during fit.
.....	13 years	.....	77, soft.	
.....	13 years	.....	100, irregular	Bites the tongue during fit.
.....	13 years	Uncle and brother epileptic.....	85, weak.	
Overwork	14 years	.....	92, weak....	Bites the tongue during fit.
.....	14 years	Father died at lunatic asylum	88, irregular	Bites the tongue during fit.
Suppressed menstruation.....	14 years	.....	Frequent and weak.	Pulse from 88 to 106.
Fear	14 years	.....	68, weak.	
Fright.....	15 years	Mother epileptic.....	96, small	
.....	15 years	Brother epileptic	88, irregular	Bites the tongue during fit.
.....	15 years	.....	69, weak	
Establishment of menstruation ...	15 years	Brother epileptic	65, weak.	

No.	Age.	Nativity.	Occupation.	State.	Nature of the Attacks.	Complications.
63	26	Virginia.....	.....	S.	General spasms.	Hemorrholds; dysmenorrhœa; limbs cold .....
64	18	New York ...	.....	S.	Petit mal and spasms	Limbs cold and painful.....
65	19	New York ...	.....	S.	Petit mal and spasms	Amenorrhœa; limbs cold .....
66	20	Connecticut...	.....	S.	Petit mal	Memory dull; limbs cold.....
67	18	New York ...	.....	S.	General spasms.	Vaginitis; limbs cold .....
68	24	Indiana	.....	W.	General spasms.	Snicidal mania; limbs cold.....
69	22	New York ...	Seamstress ...	S.	Petit mal and spasms	Phthisis; limbs cold; memory dull
70	19	New York ...	.....	S.	General spasms.	Paralysis of left arm; limbs cold ..
71	18	New York ...	....	S.	General spasms.	Amenorrhœa; limbs cold .....
72	20	Ireland	Domestic	S.	General spasms.	Dysmenorrhœa; hemorrholds ....
73	20	New York ...	.....	S.	Noct. spasms...	Dysmenorrhœa; retroflexion of the uterus; limbs very cold.....
74	18	New York ...	.....	S.	Noct. spasms...	Paralysis of right arm in the morning after fit; limbs cold .....
75	24	New York ...	Domestic	S.	General spasms.	Insanity; limbs cold..... ..
76	2	Germany	.....	S.	General spasms.	Dementia; extremities cold.....
77	25	Ireland	Domestic	S.	General spasms.	Paralysis of right arm; limbs cold
78	19	Connecticut ..	.....	S.	General spasms.	Memory dull; action of heart irregular; limbs cold .....
79	26	Ireland	Domestic	W.	General spasms.	Limbs weak and cold; phthisis...
80	23	New York ...	.....	S.	Noct. spasms...	Limbs cold and numb .....
81	28	Ireland	Domestic	W.	General spasms.	Aortic direct murmur; limbs cold
82	40	Switzerland ..	.....	M.	General spasms.	Dementia; limbs cold .....
83	23	New York ...	Teacher.....	W.	General spasms.	Amenorrhœa; body very small for her age; limbs cold and weak...
84	40	Germany	.....	S.	Rolling spasms without unconsciousness.	Dementia; syphillis; bones of nose and palate destroyed .....
85	28	Connecticut ..	Baker	M.	Noct. spasms...	Limbs always cold .....
86	22	New York ...	.....	S.	General spasms.	Fits preceded by a bad taste and choking of the throat.....
87	21	New York ...	.....	S.	General spasms.	Dysmenorrhœa; limbs cold .....
88	27	New York ...	.....	W.	Spasms on left side	Hemiplegia left side; limbs cold ..
89	30	Ireland	Domestic	W.	General spasms.	Vertigo; limbs cold.....
90	24	New York ...	.....	S.	Noct. spasms...	Leucorrhœa; limbs cold .....
91	25	New York ...	....	S.	General spasms.	Facial paralysis and converging strabismus in right side; hands cold and numb.....
92	29	Ireland	Seamstress ...	W.	General spasms.	Hallucination of hearing before fits
93	36	Canada	Seamstress ...	S.	General spasms.	Phthisis; limbs cold .....
94	28	Ireland	Cook	S.	General spasms.	Contraction in right limbs; extremities cold .....
95	36	Poland	Pedler	M.	General spasms.	Smell of smoke and numbness in left arm before fit; limbs cold ..
96	32	New York	.....	S.	General spasms.	Loud aortic direct murmur; limbs cold .....
97	36	Connecticut ..	....	S.	Petit mal and spasms	Irregular menstruation; limbs cold
98	31	New Jersey...	.....	S.	Petit mal and spasms	Painful menstruation; limbs cold.
99	28	Pennsylvania.	.....	M.	Petit mal and spasms	Paraplegia; limbs numb and cold .
100	28	Illinois	....	S.	General spasms.	Tremor and palsy after fit; limbs cold.....
101	40	New York	.....	W.	General spasms.	Dysmenorrhœa; offensive perspiration after fit; limbs cold.....
102	41	Ireland	.....	W.	General spasms.	Emphysema pulmonalis; amenorrhœa since first seized; chills and wandering of mind before fit ....

Cause.	Age of Invasion.	Hereditary Influence.	State of Pulse.	Remarks.
Rectal trouble.....	15 years	Father died paralytic .	98, irregular, weak.	
Establishment of menstruation ...	15 years	....	73, feeble.	
Undeveloped uterus	15 years	.....	96, feeble ...	
.....	15 years	.....	87, weak....	Has had only two spasms.
Onanism	15 years	....	104, weak.	
.....	16 years	.....	86, weak....	Maniacal excitement before fit.
Scarlatina.....	16 years	Mother insane.....	62, feeble.	
.. ..	16 years	.....	98, irregular	Bites the tongue during fit.
Suppressed menstruation.....	16 years	.....	100, small...	Bites the tongue during fit.
Rectal trouble.....	17 years	....	70, regular..	Fits preceded by nausea.
Impediment to menstruation....	17 years	.....	86, feeble ...	Has petit mal in day time.
Suppressed menstruation.....	17 years	.....	70, small....	Has headache on going to bed before fits.
Establishment of menstration	18 years	....	46, slow weak	
Disappointment...	18 years	.....	88, irregular	Stammers after fit.
Intestinal derangement.....	18 years	.....	94, soft.	
Stopped menstruation	18 years	.....	104, weak.	
.....	19 years	.....	90, feeble....	Bites the tongue only in nocturnal fits.
.....	19 years	....	82, feeble ...	Urine diabetic.
Grief	20 years	Mother epileptic.....	64, feeble,	
.....	20 years	.....	94, weak, intermittent.	
Difficult labor.....	20 years	.....	94, irregular	Fits seized her at birth of first child.
Syphilis	20 years	.....	74, feeble ...	Maniac after fits.
.....	20 years	.....	80, regular ..	Fits only during sleep.
....	20 years	.....	76, soft.....	Bites the tongue during fit.
Suppressed menstruation	20 years	.....	70, feeble ...	Bites the tongue during fit.
Fracture of skull ..	21 years	.....	82, feeble.	
Grief	22 years	....	80, feeble.	
Fatigue disturbing menstruation .	22 years	.....	74, feeble....	Occasionally petit mal in day time, during menstruation.
Brain fever.....	23 years	.....	87, irregular.	
Grief.....	24 years	.....	64, regular.	
Intemperance.....	24 years	Parents phthisic.....	78, weak.	
Typhoid fever.....	24 years	.....	78, soft	Bites the tongue during fit.
Fright during child birth	26 years	.....	84, feeble ...	Bites the tongue during fit.
Puerperal hemorrhage.....	26 years	.....	100, soft	Bites the tongue during fit.
Amenorrhœa.....	26 years	Mother paralytic; sister epileptic	85, feeble ...	Fits followed by vomiting.
Suppressed menstruation	26 years	.....	84, weak.	
Chills	26 years	.....	97, feeble ...	First fit after an attack of ague.
Over exertion. ...	26 years	.....	92, weak.	
Uterine trouble....	27 years	Father died paralytic..	96, weak.	
.....	28 years	.....	86, feeble ...	Cramps in the limbs, always cold.

No.	Age.	Nativity.	Occupation.	State.	Nature of the Attacks.	Complications.
103	40	New York	.....	M.	General spasms.	Systolic direct murmur at base; limbs cold.....
104	38	Ireland	Domestic	S.	General spasms.	Amenorrhœa; tremor; limbs cold.
105	37	Maine	.....	M.	Noct. spasms...	Headache previous to fits: limbs cold .....
106	30	New York	.....	S.	General spasms.	Amenorrhœa; limbs cold.....
107	36	Pennsylvania.	Housekeeper .	W.	Noct. spasms...	Maniacal excitement with fits; limbs cold .....
108	44	Ireland	Seamstress ...	W.	Noct. spasms...	Dysmenorrhœa; fits only during catamenia .....
109	32	New Jersey...	.....	S.	General spasms.	Paraplegia; limbs painful, numb and cold.....
110	32	England	.....	S.	General spasms.	Phthisis; hallucination of sight after fit.....
111	37	Ireland	Domestic ...	W.	General spasms	Extremities always cold; tremor..
112	32	Massachusetts	.....	S.	General spasms.	Temporary hemiplegia right side..
113	40	New York	.....	M.	Consci'sness not always lost ...	Hemiplegia in left side; limbs cold
114	34	Ohio	.....	M.	Noct. spasms...	Headache and vertigo before fits; limbs cold.....
115	36	Pennsylvania.	....	M.	General spasms.	Headache and deadness of right limbs after fits; limbs numb and cold.....
116	35	Maryland	.....	M.	Noct. spasms...	Uterus enlarged; leucorrhœa; limbs cold .....
117	41	Ireland	.....	W.	General spasms.	Limbs cold and numb; cramps....
118	36	New York	Domestic	S.	General spasms.	Fissure in ano; lower limbs numb and cold.....
119	38	Ireland	Domestic	M.	Noct. spasms...	Insanity; amenorrhœa; limbs cold
120	36	New York	.....	S.	Upper limbs only convulsed	Soft mitral regurgitant murmur; limbs cold.....
121	36	New York	.....	M.	General spasms.	Hemiplegia, right side after fits; limbs cold .....
122	48	New York	.....	M.	General spasms.	Dementia; hemiplegia, right side; no aphasia; limbs cold and numb
123	38	Long Island..	.....	M.	General spasms.	Prolapsus uteri; lower limbs weak and cold.....
124	41	Ireland	.....	M.	General spasms.	Sight and hearing impaired in right side; limbs numb and cold.....
125	50	Ireland	Domestic	S.	General spasms.	General paralysis; loud systolic murmur at base; pricking, numbness and cold of limbs.....
126	40	Ireland	Seamstress ...	S.	Spasms on right limb	Right arm paralyzed; limbs cold and numb.....
127	47	New York	.....	S.	General spasms	Otorrhœa; phthisis; limbs cold...
128	60	Ireland	Domestic	S.	General spasms	Phthisis; limbs numb and cold;
129	56	Germany	Domestic	S.	Petit mal and spasms	tremor ..... Paralysis of the tongue; pricking, numbness and cold in the limbs; slight impaired .....
130	19	Ireland	Domestic	S.	Spasms on right limbs	Hemiplegia left side; limbs cold ..
131	25	New York	.....	S.	General spasms.	Dementia; limbs very cold.....
132	32	Ireland	.....	W.	Incomplete unconsciousness	Carcinoma uteri; metrorrhagia; chronic bronchitis; heart enlarged.....
133	25	New York	Domestic ...	S.	Petit mal and spasms	Intellectual faculties dull; limbs cold.....
134	46	New York	.....	S.	Noct. spasms...	Hemiplegia right side; speech impaired.....
135	26	Ireland	Domestic	S.	Spasms on left limbs	Hemiplegia left side; heart's action irregular ....
136	27	Ireland	Tailoress	W.	General spasms.	Insane; slow systolic murmur at base.....
137	38	Ireland	.....	S.	General spasms.	Limbs purple and cold; dementia.
138	27	Ireland	.....	S.	General spasms.	Insanity; heart's action irregular and violent.. .....

Cause.	Age of Invasion.	Hereditary Influence.	State of Pulse.	Remarks.
Over work.....	28 years	Parents phthisie	77, weak.	
Intemperance	28 years	.....	90, feeble....	Bites the tongue in fits and is delirious after them.
Mental disturbance	28 years	.....	68, weak.	
Suppressed menstruation	28 years	.....	102, weak...	Bites the tongue during fit.
Pregnancy.....	28 years	All but the first children born epileptic...	64, slow.....	Became epileptic when she was quick with child in her first pregnancy and has been epileptic and maniacal at every successive one.
.....	29 years	Mother's cousin epileptic.....	70, weak.	
Rheumatism	29 years	Mother's sister epileptic.....	86, feeble...	Bites the tongue during fit.
Small pox.....	29 years	Mother phthisic.....	94, weak.	
Intemperance	30 years	....	68, weak.	
Dysentery.....	30 years	.....	62, very weak.	
Child birth.....	30 years	.....	94, feeble....	First fit upon post-partum hemorrhage.
Fright during lactation	30 years	.....	80, regular..	Fits preceded by sickness at the stomach.
Disappointment....	32 years	Sister died epileptic..	80, feeble.	
Child birth.....	32 years	.....	76, small....	Bites the tongue during fit.
Intemperance	34 years	Father phthisic; mother apoplectic.....	89, feeble.	
Rectal trouble.....	34 years	.....	74, small.	
Intemperance	34 years	.....	72, feeble.	
Blow on the skull.	34 years	Mother died paralytic.	100, quick.	
Grief.....	34 years	.....	86, irregular	Bites the tongue during fit.
Intemperance	35 years	Paternal grandmother epileptic.....	102, weak...	No cardiac trouble.
Uterine trouble.....	36 years	.....	72, soft.....	Bites the tongue during fit.
Miscarriage from fright..... ...	38 years	.....	78, feeble.	
Intemperance	39 years	.....	86, feeble....	Bites the tongue during fit, maniacal excitement after it.
Intemperance	40 years	.....	78, feeble.	
Otorrhœa.....	40 years	.....	68, weak....	Feels free from fit when the ears discharge.
Intemperance	45 years	.....	82, feeble....	Bites the tongue during fit.
Intemperance	48 years	Insanity in mother's side.....	80, weak.	
.....	Unkn'n.	Father insane	70, weak....	Bites the tongue during fit.
.....	Unkn'n.	.....	80, feeble.	
.....	Unkn'n.	.....	72, small.	
.....	Unkn'n.	.....	88, irregular.	
....	Unkn'n.	.....	98, irregular.	Never menstruated; no cardiac trouble.
.....	Unkn'n.	.....	87, feeble ...	Silly and demented.
.....	Unkn'n.	.....	95, irregular.	
.....	Unkn'n.	Father epileptic	82, small....	Bites the tongue during fit.
....	Unkn'n.	.....	100, irregular	No cardiac murmur.

No.	Age.	Nativity.	Occupation.	State.	Nature of the Attacks.	Complications.
139	20	Island of Cuba	Tailoress.....	S.	Petit mal,spasms	Dementia; extremities cold.....
140	24	Pennsylvania.	.....	S.	Noct. spasms....	Dementia; phthisis.....
141	(?)	Ireland.....	.....	S.	General spasms.	Dementia; extremities very cold..
142	12	New York....	.....	S.	Noct. spasms....	Dementia; limbs cold.....
143	16	New York....	.....	S.	Noct. spasms....	Mind Impaired; extremities cold..
144	(?)	Germany.....	.....	M.	General spasms.	Insanity; limbs very cold.....
145	18	New York....	.....	S.	Petit mal and spasms.....	Dementia; amenorrhea; limbs cold
146	19	New York....	.....	S.	Petit mal and spasms.	Impaired Intellect; limbs cold; spasms began at the age of 15....
147	18	(?)	.....	S.	General spasms.	Insanity; tingling of whole body
148	12	Germany.....	.....	S.	Spasms in right limbs.....	before fits; limbs cold .....
149	15	New York....	.....	S.	Petit mal and spasms.....	Dementia; hemiplegia on left side.
150	23	Long Island ..	.....	S.	General spasms.	Dementia; limbs cold.....
151	14	England	.....	S.	Spasms In upper limbs.....	Nymphomania; limbs cold .....
152	22	New York....	.....	S.	General spasms.	Dementia; left lung tuberculous; limbs cold..... .. ..
153	27	New York....	.....	W.	General spasms.	Dementia; runs before fit; limbs cold .....
154	18	New York....	.....	S.	Noct. spasms...	Drowsy and wild before fit attending catamenia..... .....
155	(?)	Germany.....	.....	S.	Petit mal and spasms.....	Dementia; amenorrhea; limbs cold
156	20	New York....	Domestic.....	S.	Petit mal and spasms.....	Insane; limbs cold and scorbutic..
157	14?	(?)	.....	S.	Petit mal and spasms	Anesthesia in right limbs, cold and weak; hearing and sight diminished in right side.....
158	35	Scotland.....	.....	W.	General spasms.	Idiocy; limbs cold and weak.....
159	31	Ireland.....	.....	W.	General spasms.	Insane; dysmenorrhœa; limbs cold
160	(?)	Germany	.....	S.	General spasms.	Imbecile; hemiplegia left side; palpitation of the heart; limbs cold and tremulous.. ..... Hemiplegia; right side; dementia; aortic direct, and aortic regurgitant murmurs.....
161	35	England.....	.....	S.	General spasms.	Insanity; general paralysis .....
162	28	England	.....	M.	General spasms.	Insanity; aortic regurgitant murmur
163	30?	(?)	....	S.	General spasms.	Manlae; Insane; limbs cold.....
164	23?	(?)	.....	S.	Petit mal.....	Intellect impaired; limbs cold.....
165	20	New York....	....	S.	Petit mal,spasms	Hemiplegia left side; speech lost after fit and very thick the rest of the time.....
166	12	New York....	.....	S.	General spasms.	Right facial paralysis and left hemiplegia.....
167	46	New York....	.....	S.	General spasms.	Dementia; maniacal excitement after fit.....
168	37	Ireland.....	Domestic.....	S.	General spasms.	Syphilis; stosis and hemiplegia left side.....
169	26	New York....	.....	S.	General spasms.	Hemiplegia right side; apbasia;
170	36	Ireland.....	.....	W.	Petit mal and spasms.....	limbs cold..... Dementia; hemiplegia left side; limbs cold..... ....
171	39	Ireland.....	.....	S.	General spasms.	Dementia; hemiplegia right side; limbs cold.....
172	51	Ireland.....	.....	S.	General spasms.	Dementia; hemiplegia left side;
173	15	(?)	.....	S.	Petit mal in day time, spasms at night.....	limbs cold..... ..... Dementia; limbs cold.....
174	18	New York....	.....	S.	General spasms.	Uterus undeveloped; never menstruated.....
175	38	New York....	.....	S.	Petit mal and spasms.	Imbecile; hands and feet cold.....
176	30	New York....	....	S.	Petit mal in day time, spasms at night.....	Fits of laugh and mania after the attacks of petit mal; intellectual faculties impaired; limbs cold...

Cause.	Age of Invasion.	Hereditary Influence.	State of Pulse.	Remarks.
.....	Unkn'n.	.....	87, feeble ...	Bites the tongue during fit.
.....	Unkn'n.	.....	84, weak....	Bites the tongue during fit.
.....	Unkn'n.	.....	94, weak....	Bites the tongue during fit.
.....	Unkn'n.	.....	99, soft	Bites the tongue during fit.
.....	Unkn'n.	.....	86, feeble.	
.....	Unkn'n.	.....	72, feeble.	
.....	Unkn'n.	.....	84, feeble.	
.....	Unkn'n.	.....	68, feeble.	
.....	Unkn'n.	.....	80, weak....	Bites the tongue during fit.
.....	Unkn'n.	.....	85, small.	
.....	Unkn'n.	.....	102, soft.	
.....	Unkn'n.	.....	70, irregular	Was blind for a year.
.....	Unkn'n.	.....	80, small.	
.....	Unkn'n.	.....	70, natural..	Bites the tongue during fit.
.....	Unkn'n.	.....	89, irregular.	
.....	Unkn'n.	.....	95, weak.	
.....	Unkn'n.	.....	74, feeble ...	Bites the tongue during fit.
.....	Unkn'n.	Sister epileptic	82, small....	Mental facultics deficient.
.....	Unkn'n.	.....	87, weak.	
.....	Unkn'n.	.....	92, irregular.	
.....	Unkn'n.	.....	95, irregular	Dysphagia after fits.
.....	Unkn'n.	.....	92, weak and small.	
.....	Unkn'n.	.....	78, small.	
.....	Unkn'n.	.....	88. small....	Maniac after fits.
.....	Unkn'n.	.....	75, feeble ...	Bites the tongue during fit.
.....	Unkn'n.	.....	68, irregular	Always silent and apathetic.
.....	Unkn'n.	.....	80, weak....	Limbs hyperæstbetic before fit.
.....	Unkn'n.	.....	95,very weak	Died the day after coming into the hospital.
.....	Unkn'n.	.....	98, irregular	Bites the tongue during fit.
.....	Unkn'n.	.....	88, feeble ...	Aura from left hand.
.....	Unkn'n.	.....	90, weak....	Had fits long before paralysis.
.....	Unkn'n.	.....	72, small.	
.....	Unkn'n.	.....	79, feeble ...	No signs of cardiac disease.
.....	Unkn'n.	.....	85, weak.	
.....	Unkn'n.	.....	68, feeble.	
.....	Unkn'n.	.....	76, irregular	Has had petit mal; imbecile.
.....	Unkn'n.	.....	70, feeble ...	Bites the tongue during fit.
.....	Unkn'n.	.....	80, irregular.	

Nota.—Cases 8, 140, and 87, were: the two first negroes and the third a mulatto.

In ascertaining the ages of invasion we may see by referring to the annexed statement, that the maximum corresponds with the period of adolescence, namely, from fourteen to twenty-five years. This result upholds those yielded by the calculations of Moreau de Tours, embracing 995 cases;* Musset, 307 cases;† Sieveking;‡ Reynolds;§ and Girard de Cailleux.‖ It seems, therefore, as generally acknowledged, that the first appearance of epilepsy must be materially influenced by puberty, or rather by adolescence, at which age, in the words of Girard de Cailleux, the storms of puberty burst out while the organism seized with the seminal fever, described by Bordeu, turns to be the theatre of a new evolution from the first dawning day of puberty until growth is fully achieved.

It may be observed that among 168 of my patients, independently of those from the Hospital for Epileptics, the males bear a greater proportion at all periods, excepting from 6 to 13 years, when their number is equal to that of the females.

Periods.	Males.	Females.	Periods.	Males.	Females.
0–5	23	17	35–45	12	2
6–13	12	12	45–58	6	1
14–25	23	20	Unknown,	"	3
25–35	20	17		—	—
Total,				96	72

* Del Etiologie de l'Epilepsie. Mémoires de l'Academie de Médicine de Paris, 1855, Tome XVIII.

† Marié traité des Maladies Mentalis. Paris, 1862, p. 523.

‡ Op. cit., p. 109.

§ Op. cit., p. 126.

‖ Op. cit., p. 58.

In the statistic of Sieveking at every one but the last decennial period from birth to 68, the males are prevalent, and the same happens with the table of Girard de Cailleux, in which there is, however, a larger amount of females set down from 12 to 20 years. But the superiority of the male number displays itself more conspicuously in the combination I have analyzed, with the object of gaining more extensive information, comprehending the tables of Sieveking, Reynolds, Girard de Cailleux, and my own. This general statement foots on the whole 639 cases, near equally divided among both sexes; there being 323 males to 316 females. The grand total in this estimate, notwithstanding the unusual proportion of females arising out of my table, agrees with the results specified in the official statistics of death from epilepsy in England and Wales, and in the United States. The fact is worth noticing, for it evinces how discrepancy between partial statistics always corrects itself when they enter into a larger calculation, and how cautious must we be in not placing an absolute reliance upon such individual returns. Let me say, that in reckoning the comparative periods in the table below, I have made them to correspond with the first and second infancy, adolescence, adult and old ages, as the division appears the more natural, and, with comparatively trivial difference, adapts itself to those adopted by the authors whose tables I quote.

Table A.

Periods.	Sieveking.			Reynolds.			Girard dec.			Echeverria.			Grand Total.			Comparative Periods.		
	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.	Males.	Females.	Total.
From 0 to 5 years,	16	13	29	2	6	8				3	3	6	36	27	63	54	46	100
" 0 to 10 "				5	5	10							16	13	29			
Under 5				3	1	4							2	6	8			
6 — 10							6	2	8	19	27	46	19	27	46			
6 — 13													3	2	5			
11 — 12													6	2	8			
Under 12													23	21	44	51	51	102
11 — 20													11	18	29			
12 — 20							11	18	29	32	40	72	32	40	72			
13 — 15				11	8	19							3	4	7			
14 — 25				3	4	7							7	7	14			
16 — 17				7	7	14							26	12	38			
18 — 20				5		5	26	12	38				5		5			
20 — 25													7	4	11			
21 — 30			11	5	3	8				26	28	54	26	28	54			
25 — 35		4					22	11	33				5	3	8			
26 — 47				5		5							22	11	33			
30 — 40		2	7				12	6	18	8	6	14	5	2	7			
31 — 45													8	6	14			
35 — 50		4	9				3	2	5	9	1	10	12	6	18			
40 — 50													5	4	9			
41 — 58													9	1	10			
45 — 60													3	2	5			
50 — 60		3	4				3	2	5	3	..	3	1	3	4			
51 — 68													3	..	3			
53 — 66													3	2	5			
Over 60				1	..	1							1	..	1			
69 — 70				3	2	5							12	53	65			
Unknown.							8	4	12	1	47	48						
Total,	57	47	104	45	36	81	91	57	148	130	176	306	323	316	639	92	61	153
																11	7	18

Reynolds asserts: "That there is nothing in the sexual organization of individuals which, from ten to twenty years of age, renders one or the other sex more prone to epilepsy; but, that there is some sexual character which renders females under ten years of

age more subject than males to epilepsy; and that there is something which renders males after twenty years of age more likely than females to become epileptic."* The partial tables of Sieveking, Girard de Cailleux, my own, and the above combined one, do not uphold the inferences just quoted. They manifest on the contrary, that, at all ages, males are more prone to epilepsy than females, the only exceptional period in which females rank higher, being solely observable in Girard de Cailleux's table, but at a time in which Reynolds assumes, that there is nothing rendering one or the other sex more prone to epilepsy.†

It is obvious, by thus extending the estimate over a larger amount of cases, that there is no ground to advance that, epilepsy is developed earlier in females than in males, as Sieveking‡ and Reynolds§ have been led to suppose upon their respective estimates. It seems as though from birth to twelve years, both sexes were equally subject to the disease, although in

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* Op. cit., p. 127.

† In the valuable article on Epilepsy, in a System of Medicine, London, 1868, vol. II, p. 254, Reynolds has published the following more extensive table, which, in every respect, agrees with the above remarks:

Age at Commencement.	Males.	Females.	Total.
Under 10 years, - - - -	10	9	19
Between 10 and 20 years, - - -	66	40	106
Between 20 and 45 years, - - -	25	20	45
Over 45 years, - - - -	1	1	2
	102	70	172

‡ Medico-Chirurgical Transactions, London, vol. XL, p. 158.

§ Op. cit., p. 125.

the period of first infancy, there is a slight increase in the male number. The considerable amount of females in the fourth column of the combined table ought to have strengthened the conclusion of the two distinguished English authors, and so much the more, as before the age of twelve the statistic of Girard de Cailleux adds little to the gross results, therefore, mainly derived from cases of Sieveking, Reynolds and my own. Consequently, I should deem the total in question as the most nearer to the truth, and should assume thereon, that: during infancy both sexes are equally subject to epilepsy, although during first infancy, males are more than females liable to it. This, of course, is a conclusion that I cautiously lay down, ready to modify it whenever more extensive calculations will show its inaccuracy. It further strikes in the preceding table that the invasion of epilepsy gradually augments up to adult age, to decrease thereafter; the lessening being no more rapid in the female than in the male number. The total in adult age does not fall off in any great degree from that of adolescence, and it is at that particular age that intemperance takes an important share in thus maintaining the number, for it contributes among lower and higher classes to augment, in a considerable proportion, the total of adults and old people affected with epilepsy. The production of epilepsy is in no evident way influenced by the critical time or menopausis, for we fail to discover any excess, over the preceding, in the return belonging to this period in the

female column. Not so, however, with the age of first appearance of menstruation, in which the total of female swells over that of any other period; but to this important subject I shall revert when discussing the accidental causes of epilepsy.

I have separately grouped the hereditary influences specified in eighty of the cases which have come under my observation. The information in every instance has been as accurate as could be wished, for it principally derives from the patient's family, and, unless sufficiently proved, has never been accepted from the patients themselves. I have, as much as possible, extended my inquiry to every affection under which the parents might have labored; this I have done, not imagining that epilepsy could be assigned hereditarily to every variety of preexisting affection, but to ascertain the proportion which intemperance and phthisis bore to other kinds of hereditary causative conditions. In regard to the predisposition descending from affections of the nervous system, unlike to epilepsy in the parent, *a priori* it can be scarcely contradicted in a disease so intimately connected with nervous derangements generally. Lucas, Moreau de Tours, Baillarger, Morel, Herpin, Guislain, Griessinger, Briquet, Trousseau, and several more, have adduced facts establishing such successive transformation of every kind of neurosis into epilepsy, insanity, hysteria, locomotor ataxy, paralysis, etc., from parent to offspring. Wherefore, I will not waste time in accumulating evidence to that abundantly supplied by these dis-

tinguished authorities. I am aware that among as highly standing advocates of contrary views, Parkes * and Sieveking † discountenance the admission of every variety of nervous affection as a hereditary predisposing cause, because of the vague data to establish the fact, and the necessity of including in the calculation, every exhausting or debilitating disease. While entertaining all respect for this opinion, I feel obliged to say that, it by no means disproves the occurrence of such predisposition, but only renders more notorious the difficulties misleading us in its elucidation. Indeed, we may well affirm that any malady or degeneracy in offspring does not proceed from some analogous or equal organic derangement ingrafted on the parents, for, even were they actually exempt from any ostensible abnormity, the evidence would weigh little on the question. There is to be accounted for, prior to the affirmation, the alternate existence observed through the hereditary continuance of organic attributes, whether physiological or morbid, by which disease not unfrequently skips one generation to spring up anew among members of the next, just as in the pedigree of animals we discover special characters wanting in one progeny and thereafter displayed by individuals of the successive ones. And then again, there are beside latent taints, influences surrounding the parent very difficult to suppress, and the part assignable to these efficient

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* British and Foreign Medico-Chirurgical Review, April, 1853.

† Op. cit., 113.

elements must be previously discriminated, whether we undertake to uphold or to contradict, with our estimate, the hereditary transmission of epilepsy by other pre-existing neuroses in the parents. Therefore, the safest way to investigate the subject consists in starting from offspring to ascendant, as Trousseau and Briquet have done, and is suggested by Rance and Boudin,* to ascertain the true influence of marriages of consanguinity on offspring. By such a course we undoubtedly arrive at the conclusion that, for a parent to transmit epilepsy to the offspring, it is not necessary that he should himself present the spasmodic neurosis, for any other one propends to same results. We should not exact an analysis of every possible case; the ability of the experimenter, as Jules Simon says, consists in seizing the moment when the number of facts suffices to establish the law. But, the examples of this variation in the hereditary transmission of neuroses are notorious; thus: among 351 hysterical women, whose families amounted to 1,103 individuals—430 males and 673 females, Briquet found among ascendants and collaterals: 214 hysterical, 13 epileptic, 16 insane, 1 with delirium tremens, 1 with paraplegia, 3 somnambule, 14 with convulsive disease, and 10 with apoplexy. Upon the whole, 272 cases of nervous affections out of 1103 individuals, or nearly 25 per cent., and whereas there were mothers and

* Bulletin de la Société d'Anthropologie. Paris, 1863. Tome IV, pp. 613, 624 and 674.

sisters, whose condition could not be ascertained, and some of them must necessarily have been affected with a similar nervous disease, we may raise the amount to the round proportion of 25 individuals attacked with affections of the encephalon out of 100 parents, or nearly one-fourth.* Todd reports the case of a hysterical woman, whose paternal grandfather and paternal uncle had fits, and she was the mother of nine children, eight of whom died, seven of them in convulsions.† With these, and more examples which can be adduced, we are forced to admit, as Falret sets it down in terms more competent than mine: "Upon the whole, such general influences, after having, in the beginning, impressed the nervous system of ascendants in a benignant manner, increase and aggravate their effects by inheritance. Within the range of the nervous system and of the moral, there is a successive bending from purely nervous diseases, such as hysteria, epilepsy, hypochondria, or the proteiform neuroses, to morbid eccentricity of character, to naturally or morbidly aggravated evil propensity, that is, to the various forms of moral or instinctive insanity, or further, to an intelligence either precocious and soon extinguished, or very partially and very unevenly developed. These mixed conditions propend, through uninterrupted series of pathological states, towards distinct forms or varieties of

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* De l'Hystérie. Paris, p. 82.

† Op. cit., p. 471.

mental disease and, ultimately, entail upon the offspring different degrees of imbecility and idiocy—that is to say, extinction of intelligence. In the sphere of other organs the same deleterious influences follow a parallel course in each succeeding generation, commencing in lessening of stature and slight local or partial abnormities, until terminating in more and more extensive organic malformation, and lastly in complete extinction of the race by premature death of children, who though often born in great number, nevertheless die all at a young age."* No better delineation could be presented of the morbific hereditary transmission which takes its principal source in diseases of the nervous system, a subject again made evident in high degree by Morell in his admirable work on Degeneracies of Mankind. Moreover, this peculiar variation which accompanies the hereditary transmission of neuroses, is not unprecedented in the pathogeny of nervous affections. We know, and Brown Séquard has pointedly remarked: "That the same peripheral cause of irritation acting on the same centripetal nerve may produce the greatest variety of effects, including every functional nervous affection or disorder."† In like manner, we may conceive that, the same nervous diathesis might originate a variety of peripheral effects or symptoms, under the influences acting on each

* De la Consânguinité. Paris, 1865, p. 37.

† On the Diagnosis and Treatment of Functional Nervous Disorders. Philadelphia, 1868, p. 15.

individual case, and which are very difficult, if not impossible, to discriminate. While on this subject, I could not pass unnoticed the opinion expressed by Trousseau in one of his most interesting lectures on epilepsy, and previous to his giving some unmistakable instances of hereditary taint as a powerful predisposing cause of the disease. "Hereditary taint," says he, "has certainly a great influence on the production of epilepsy, and I hardly understand how trustworthy authors can have doubted such a fact, which has been accepted by the generality of practitioners. They may have been misled by the circumstance that disorders of the nervous system assume the form of epilepsy in some individuals, and in others of phenomena of an apparent different character. This transformation of nervous affections into one another is a vast subject, which I can not consider now; but if you question your patients scrupulously — if you carefully inquire into their previous history, you will, in many cases, discover, either in their direct or collateral relatives, symptoms analogous to those which they themselves present, or mental alienation in one of its various forms, or mere eccentricities of character or of manner; or, again, disturbances of innervation characterized by strange symptoms, by peculiar nervous phenomena, which indicate an unfortunate predisposition transmitted from generation to generation."*

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* Lectures on Clinical Medicine. Translated by P. Victor Bazire, M. D. London, 1867, p. 79.

The question of hereditary taint as a predisposing cause of epilepsy, suggests itself under three aspects:

1st. The reproduction of epilepsy directly traceable to the same affection in the parent.

2d. As a modal diversity or transformation of pre-existing neuroses in the ascendants, entirely different from epilepsy itself.

3d. Through agency of a systemic, but not essentially nervous, derangement in the parent, extending its injurious effects to the offspring. All these circumstances involve morbific influences of various kinds equally detrimental to ascendants and descendants, sparing neither of them, and transmissible in their effects through direct or indirect line from generation to generation. Yet, hereditary predisposition may further be traceable to a source quite innocuous to the parents themselves, but highly disastrous to offspring, in marriages of consanguinity which, on this account, ought to be separated from the above predisposing causes.

Let us now analyze the following eighty cases in reference to each of the foregoing points: I may premise that I have relied on facts, not statements, to record the agency of hereditary influences. I do not mean by this to assure for one moment that in all cases, and particularly with those belonging to the third group, epilepsy was the immediate result of the organic disease exhibited by the parents of the patient. But, it is not improbable that, such systemic derangement, by lessening nutrition generally, may bring about the interstitial changes of the medulla apparent in epilepsy.

Table showing the Hereditary Influences, as recorded in 80 out of 306 cases of Epilepsy.

		Males.	Females.
Ascendants.	1. Mother epileptic,	2	4
	2. Mother epileptic and father phthisic,	1	1
	3. Mother epileptic and phthisic, father inebriate,	..	1
	4. Father epileptic,	2	1
	5. Paternal grandmother epileptic,	..	1
	6. Maternal grandmother idiotic. (Patient's sister epileptic),	1	..
	7. Mother's and father's brothers epileptic,	1	..
	8. Father's brother epileptic,	1	..
	9. Father's sister epileptic,	1	..
	10. Father's cousin epileptic,	..	1
	11. Mother's brother epileptic,	1	.
	12. Mother's brother and cousin epileptic,	..	1
	13. Mother's sister epileptic,	..	1
	14. Mother's cousins epileptic,	..	1
	15. Mother's relatives epileptic,	..	1
	16. Mother insane,	..	3
	17. Mother maniac,	1	..
	18. Mother imbecile,	1	..
	19. Mother intemperate (in one case two of patient's children died with fits),	2	2
	20. Mother paralytic (in one case patient's sister epileptic),	..	3
	21. Mother and patient's brother died apoplectic.	..	1
	22. Father insane,	..	1
	23. Father intemperate,	2	1
	24. Father paralytic (in one case [male] patient's brother epileptic),	2	4
	25. Father with cerebral softening,	..	1
	26. Father paralytic and inebriate (patient's brother idiotic),	1	..
	27. Father and mother inebriate,	1	..
	28. Father died in a lunatic asylum,	..	1
	29. Parents nervous,	..	1
	30. Insanity on mother's side (first cousins),	1	..
	31. Brother of maternal grandmother insane,	..	1
	32. Parents consumptive,	1	4
	33. Father consumptive,	1	1
	34. Father consumptive, mother apoplectic,	..	1
	35. Mother consumptive, father cardiac disease,	..	3
	36. Father with cardiac disease,	..	2
	37. Father's family consumptive,	1	..
	38. Uncle (maternal) and brother epileptic,	..	1
	39. Brother epileptic,	1	2
Collaterals.	40. Sister epileptic,	2	2
	41. Sister and cousin epileptic, none of the parents,	1	..
	42. Brother insane,	1	..
	43. Brother and cousin idiotic,	1	..
Consanguinity.	44. Parents first cousins; patient's brother idiotic in one case, with inguinal hernia in the other,	..	2
		30	50

We may see at a glance a greater hereditary predisposition in the female than in the male side: 30 of 130 males exhibiting hereditary taint, or 23.07 per cent, and 50 of 176 females, or 28.40 per cent. In the majority — 24 cases, epilepsy itself pre-existed among ascendants; 16 times the taint descended from the mother's side, whereas only in 7 from the father's. The difference in these proportions, and the greater frequency, in the remaining cases, of the disease likewise conveyed through the mother's line, shows that epilepsy is more frequently transmitted on the mother's than on the father's side, as it further happens with insanity. Among French authors, Trousseau expresses no special opinion on the subject: of the five examples fallen under his observation, cited in the above lecture, only one shows the hereditary taint traceable to the mother. It is so important and so briefly sketched that I transcribe it here: "A gentleman, now eighty-eight years old, was affected, at the age of sixty-four, with melancholia, of which he is at present perfectly cured. He had three children, two sons and a daughter. The eldest son is of a melancholic temperament, but of perfectly sound mind; the second was affected with locomotor ataxy, and died mad. A son of the latter, at present thirty years old, is as yet of sound mind, but has a child who is an idiot. The daughter, who is devoid of intelligence, and is, besides, somewhat strange in her ways, has had two sons, the eldest of whom died insane and paralyzed, whilst the younger one is almost idiotic.

This gentleman had also a *sister* who became mad at the age of thirty. This lady had a son and a daughter, the first from infancy has suffered from night blindness, and is now afflicted with *epilepsy*, the second was amaurotic, and died insane, leaving also a son, who has already given proofs of a notable impairment of the intellect."* The three generations of these two families—were other examples wanting—should establish in plain manner the variation of neuroses on being transplanted from parent to offspring. Esquirol† states that, upon investigation of the female epileptics at Salpêtrière, he met with epilepsy more frequently transmitted on the father's than on the mother's side, the reverse taking place with insanity. I may further notice, in regard to insanity, that; among four hundred and fifty-three cases tainted with hereditary predisposition, Baillarger traced the maternal influence prevailing in a third of the cases, and under such circumstances the female offsprings were the most liable to contract the disease.‡ From Reynolds's estimate§ "it is apparent that hereditary, or at all events family, proclivity to nervous disease is present in 12 of 38 epileptic individuals, or in 31 per cent; and further that the tendency is equal in the two sexes, 7.2 (males) and 5.16 (females) being each

* Op cit., p. 80.

† Des Maladies Mentales. Paris 1838. Tome I, p. 306.

‡ Morel — Traité des Maladies Mentales. Paris, 1680, p. 117.

§ Op cit., p. 124.

equal to 31 per cent. If we exclude the latter series in which collateral relations exhibited nervous derangement, we find that there were still 16 per cent, in whom the disease might be fairly considered as hereditary. To my own mind, however, the evidence of hereditary or of family taint is as evident in the second series as in the first; and I am therefore, led to believe that a hereditary tendency to epilepsy is much more common than it is generally represented to be by recent writers upon the subject. Esquirol stated, that epilepsy was more frequently transmitted on the father's than on the mother's side; but so far as my own cases show, the reverse of this proposition is much nearer to the truth." Sieveking observes "that epilepsy prevailed among members of the family of his patients in 13.4 per cent of his cases;" * but no allusion is made to the proclivity to, or more frequent transmission of, the disease in either sex.

The returns of my table contradict the opinion of Esquirol more completely than those of Reynolds, whose results mine fully corroborate, confirming the greater frequency with which epilepsy is transmitted on the mother's than on the father's side. Epilepsy independent of other trouble, as already advanced, was specified in 24 of 78 epileptics whose relatives were affected with some nervous or other disease. The proportion which such number bears to the whole cases reviewed is, as 24 out of 306, or 7.84 per

* Op. cit., p. 112.

cent. The percentage within a fractional difference, results almost the same with both sexes; 10 of 130, or 7.69 per cent among males, and 14 of 176, or 7.95 per cent among females.

Insanity in the antecedents is recorded eight times; six on the mother's side, and two on the father's, including the case where the patient's father died at a lunatic asylum. Adding those two cases in which respectively the maternal grandmother was idiotic, and the mother imbecile, mental disease will figure in 10 of 78, or in 18.82, per cent, of cases exhibiting any hereditary taint, and in 3.26 per cent of the whole 306. The ratio is among males as 4: 130, or 3.07 per cent, and among females as 6: 167, or 3.40 per cent. By a curious coincidence paralysis occurred as many times as mental disease, but oftener on the father's than on the mother's side, in a ratio as 7: 3; thus yielding a percentage, of 5.38 among males, and 1.70 among females. Grouping the cases of mental disease, and paralysis, with the other three of cerebral softening, apoplexy and nervousness, they return a total of 23 nervous affections. Their percentage of 7.50 differs a trifle from that of epilepsy 7.84; but not so with the corresponding proportion in either sex, for it amounted to 11 out of 130 males, or to 8.46 per cent, and to 12 of 176 females, or to 6.81 per cent, which proportions are different from those above noticed with epilepsy in either parental side.

The predisposing agency of intemperance calls for special attention, as it is certainly true that this vice

of the parent entails its pernicious results upon the children to the third and fourth generations. Morel has investigated the influence of alcoholic intoxication in the parent, and although doubtless idiocy and mania are usually exhibited by the surviving offspring, in most cases, however, the child perishes after birth, or is attacked with epilepsy precursory of stupidity and palsy. Morel reports, among other instances, that of a habitual drunkard with seven children, of whom two died with convulsions, a third was insane, the fourth melancholic, the fifth irritable and misanthropical, the sixth — a daughter — hysterical, and finally the seventh nervous, weak, and low spirited.* The same author, after narrowly watching the dreadful effects owing to intemperance in one family, from first to fourth generation, thus sketches them :

First generation — Immorality, Depravity, Intemperance, and Sottishness.

Second generation — Hereditary Drunkenness, Maniacal attacks, General Paralysis.

Third generation — Temperance, Hypochondriac tendency, Lypemania, Systematic ideas of Persecution, Homicidal Proclivities.

Fourth generation — Intelligence hardly developed. First maniacal attacks at sixteen years. Stupidity leading to idiocy; and lastly, probable extinction of the race.† Comments are needless upon this truthful and

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* Des Dégénérescence del Èpècc Humaine. Paris, 1857, p. 123.

† Op. cit., p. 125.

sad picture of intemperance potency to undermine so deeply our frame, and to contribute in such a degree to physical and moral degeneracy of mankind.

Demaux* asserts that: among thirty-six epileptics that he examined during the course of twelve years, and whose history was well known to him, five were conceived while the father was intoxicated. He has further observed two children affected with congenital paraplegia in the same family, when, from the distinct statement of the mother, conception took place during drunkenness. Lastly, the same cause was discovered in the case of a young male lunatic, aged seventeen, and in an idiot aged five years.

In addition to the foregoing, I might cite these examples from my own practice. A consumptive, of daily intemperate habits, had during the four last years of his life repeated attacks of delirium tremens, melancholia, wakefulness, etc. This man, whose wife and other children were healthy, had during that period two children, who died with fits, when respectively three and five months old. In the case of a lady, troubled with vertigo, hallucination, choreic movement, and other nervous symptoms, induced by secret unsuspected potations, and on whose obscure derangement I was lately consulted, the youngest child, born since the insane impulse existed, has been at different times seized with convulsions and is now

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* Comptes Rendus del'Académie des Sciences. Paris, 1860, Tome LI, p. 576.

paralyzed. The evil agency is again obvious from first to third generation in Case 18 of table of female epileptics, where the mother, a hopeless victim of intemperance, begot a daughter epileptic, who herself gave birth to two children carried away by fits. No less suggestive is No. 9 of the same table, in which the mother epileptic and phthisic and the father intemperate, engendered a daughter epileptic imbecile.

The propagation of epilepsy by dissimilar nervous disease rooted on the patient's parent, is witnessed in cases Nos. 6, 20, 24, and 26, in table of hereditary influences; for with the three first, either a brother or a sister of the patient was epileptic, and in the fourth case a brother was idiotic. Therefore, these instances plainly afford corroboration of paralysis in the father or mother, or idiocy in the grandmother, respectively playing a cardinal part in the production of epilepsy among the children. The power of hereditary taint is considered so capital by Trousseau, that on the evidence of trustworthy authors he further states: "individuals born of a second marriage between a perfectly healthy woman, and an equally healthy man, have been seized with the same complaint to which the woman's first husband was subject."* Vidal de Cassis, holds that the

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* Op. cit., p. 82. For those interested on the subject I join the abstract of cases culled by Boudin in his valuable, elaborate paper, "*Danger des Unions Consanguines, et Nécessité des Croisements dans l'Espèce Humaine et parmi les Animaux.* Annales d'Hygiène Publique et de Médecine Légale. Juillet, 1862, 35e numéro, pp. 27, 28, 29. Some of these examples are quoted by Trousseau in sup-

impression of syphilitic semen affixed upon the ovary, is not confined to the first conception, the following offspring may be again contaminated, and that, after copulation with a different husband. This view rests on, and is illustrated by, this case observed at Lourcine Hospital. A woman, whose first husband suffered from very obstinate syphilis, had a child dying with most unmistakable signs of syphilis. This very woman after her husband's death, married again a man

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port of the above assertion. Dr. Nott. Types of Mankind, 4th edit., p. 396, says: Instances are cited, where a negro woman bore mulatto children to a white man, and afterwards had by a black man other children, who bore a strong resemblance to the white father, both in feature and complexion. The case, is further added, of an Englishman who had by a negro woman six children, of whom the youngest ones bore more and more the European feature and complexion. Reil states that, a woman, who was slapped at the face during her first pregnancy, had therefore several children with a peculiar stain in the cheek. Gratiolet reported the case of a widow, married again to a man perfectly formed, and who gave birth to a single child affected with torticolis, as was the first husband. Simpson, of Edinburgh, asserts that a young woman, born of white parents and who had a mulatto brother born before marriage, showed unmistakable traces of black blood. According to Dr. Ogive, a woman at Aberdeen, had married twice, and had borne children both times. All of them were, as her first husband, scrofulous, although she herself and her second husband were perfectly free from all scrofulous taint. Dr. Dyce knew a creole woman who had two children of fair complexion by an European, and subsequently, married to a creole, gave birth by him to two children resembling, both in feature and complexion, the first husband. Vanina reports to have known a woman with an adulterous child resembling her absent husband, and Aldrovandi cites a similar example. This resemblance of the adulterous son to the reputed father was well acknowledged by the ancients, and hence the saying: *Filium ex adultera excusare matrem à culpa.*

perfectly healthy, she herself being also healthy, or at least, without evidence whatever of syphilis. Four years after the first marriage, and eohabiting only with her husband, she gave birth to a syphilitie child.* This case bears strong analogy to that of Ogive, quoted by Boudin. In eonneetion with sueh unaccountable impression left by first pregnaney on the generative organs, MeGillivray asserts, and I eopy it from Boudin, that if a thoroughbred beast is served by a different stoek, the fecundated dam beeomes thereafter eross-bred, her blood remaining for ever tainted from the very faet of her cross-breeding with a different stoek. The ineompeteney of the aboriginal females of New Holland to proereate with native males, after having children by an European, is reported by H. Thomson; and Count Strzelecki writes, that on his travels through Ameriea and Oceaniea, he remarked among the Hurons, Seminoles, Araueans, Polynesians, and Melanesians, that the female savages ceased to be fruitful with men of their own raee, after intereourse with the whites, although still continuing eapable of proereating with the latter.†

To elass phthisis among the diseases of parents eapable of superindueing epilepsy in offspring, may at first sight appear an altogether untenable opinion. However, a little eonsideration will manifest that it is

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* Traité des Maladies Vénériennes. Paris, 1855, p. 539.

† Edinburgh Monthly Journal of Med., etc., Oct., 1851, and Physic. Description of New South Wales and Van Dieman's Land. London, 1845, p. 346.

perfectly consistent with the tendency of lesions of the medulla oblongata to induce pulmonary tuberculosis, pointed out in one of the preceding pages. The transformation may seem the more doubtful, since the phenomenon did not attract Louis' attention in his admirable researches on phthisis; but the same Louis, Andral, Briquet, and others, have found that all phthisics are not begotten by phthisical parents. This, of course, evinces that there are other causes, outside of hereditary predisposition, originating phthisis, and does not exclude that the disease might eventually be the outgrowth of some neurosis — insanity, epilepsy or any other nervous affection — of the parent, capable itself, of successive transformation into phthisis. The estimate of Briquet, to ascertain the condition of health among antecedents of 351 hysterical women, returns 9 fathers and 13 mothers phthisical; phthisis did not exist in the father or mother of 167 women, indiscriminately taken from 25 years to old age; and in the former cases its proportion was higher than that of epilepsy, present four times in the patient's father, and once in the mother. Scrofulous or cancerous cachexia was not traced in the antecedents of the epileptics I have treated, but I have met with a very manifest gouty or scrofulous hereditary diathesis, in cases of paralysis and epileptiform convulsions. Among the ablest authors, Lugol, Moreau de Tours, Baillarger, and others, hold that scrofula, tubercle, or cancer of the parents may induce in children, a predisposition to insanity, epilepsy, or any other neurosis.

This view is sustained by evidence, perhaps lacking of the absolute conclusiveness granted to it, by the above distinguished advocates; being without sufficient data to judge the question, I will not engage myself in speculations concerning it. In my belief, hereditary taint, whether of nervous or cachectic disease, is not an unimportant predisposing element of epilepsy, but I am not disposed to uphold extreme views on the subject. As regards phthisis, it existed without other disease, eight times in the parents of the 306 epileptics here contrasted, namely: in 2.61 per cent of the cases. Three times the mother had cardiac disease, and the father, phthisis; once we find, in addition to a consumptive father, the mother apoplectic or epileptic; or again, the mother epileptic and phthisical while the father was a habitual drunkard. By referring to the table of female epileptics we will see that five among them showed symptoms of phthisis, as well as their parents. It is proper not to overlook that in three cases in which, respectively, the mother was epileptic, insane, or paralytic, the daughter had epilepsy and phthisis. Cardiac disease occurred in the ascending line from the female epileptics only twice, on the father's side, and reference has already been made to cardiac disease in the father, and consumption in the mother. Finally, in one instance, the mother seized with frequent severe attacks of malarial fever, gave birth to a child "born with fits," and epileptic thereafter.

Whether there is any positive relation of cause and effect in the cases of phthisis and epilepsy just laid down, I can not affirm. Resting on the well known experiments of Schiff,* who generated pulmonary tuberculosis, by dividing the ganglia of the pneumogastric in rabbits, Van der Kolk very significantly observes: "Since, as experience has taught me, we so often see pulmonary consumption occur in families, some members of which are affected with insanity, so that I have often seen that children who were spared from insanity, were the victims of phthisis, and that the two diseases frequently alternate with one another, or coexist, the question has often suggested itself to me, whether we might not admit the existence of a phthisis excentrica, namely, one whose first cause is to be sought in an irritated condition of the medulla and the vagus, such as so frequently occurs in phthisical subjects. It must remain for further investigations to confirm this view. I shall only add, that in such cases we often observe signs of spinal irritation in the neck, and that cupping and blistering often prove eminently useful."† As there is such an analogy of the original conditions upon which epilepsy and phthisis may depend, and that, from one to another generation, insanity may transform itself into epilepsy or phthisis — might not, under yet undefined

* Rose und Wunderlich, Archiv. für Heilk., Phys. C. Jahrg., 8 Heft, pp. 769, *et seq.*

† Atrophy of the left Hemisphere of the Brain. New Sydenham Society. London, 1861, p. 170.

circumstances, phthisis also be capable of the same curious variation, and epilepsy be the form assumed by the inherited condition disturbing the medulla oblongata?

Concerning the predisposition to epilepsy arising from disease of the heart in parents, the facts are also too isolated to decide upon any conclusions. The relation — if existing — seems not an immediate one. The tendency of cardiac disease is, by interfering with the impulsive force of circulation, to induce in the generality of cases cerebral hyperæmia, which leads to comatose rather than to epileptic symptoms. There is, however, an important circumstance in which cardiac disease may originate epileptiform convulsions, upon embolism of the cerebral arteries by migratory clots detached from the valves or cavities of the heart. In this case, moreover, convulsions are the accompaniment of the paralytic affection thus originated and, to which rather than to the cardiac disease itself, I do not hesitate to say that the outgrowth of epilepsy in children begot by individuals in such manner affected should be ascribed. I understand that cardiac disease impairing cerebral circulation and nutrition, may cause a nervous affection of the parent which will be the one to transmit the epileptic predisposition to offspring; or that the paroxysm might supervene upon a deficient supply of blood to the brain, as in that curious example of epilepsy attended with hemiplegia and jaundice, reported by Stokes,* and in which, there was

* Diseases of the Heart and Aorta. Dublin, 1853, p. 206.

an enormous dilatation of the left ventricle, with great enlargement of the mitral orifice. As Prichard and some later writers explain the phenomena of epilepsy by a determination of blood to the brain, and as hypertrophy of the heart has been looked upon as a predisposing cause of cerebral apoplexy, by Le Gallois, Corvisart, Bouillaud, Ménière, Rokitansky, Leubuscher, and others, I will briefly remark, that this influence, already rejected by Rochoux* and Todd,† is further proved of no account by the researches of Eulemberg.‡ and Bouchard.§ These latter authors have confirmed, as previously advanced by Todd, that, in by far the greatest number of cases of cerebral hæmorrhage, the predisposing cause is degeneration of the large and small cerebral arteries; hypertrophy of the heart being, therefore, much less frequently associated with apoplexy, than arterial degenerations.

The evidence afforded by the ten cases in which the collateral branches of the patient's family showed epilepsy, or some other nervous disease, is to me, strong, if not conclusive indication, of a hereditary taint, probably descending through indirect relatives, and consequently, unaccounted for by the patient. In that case in which the patient's sister and cousin were epileptic, and the parents exempt from nervous affec-

* Areh. Gén de Méd. Tome, XI, 1826.

† Op. cit., p, 116.

‡ Archiv. fur Pathologisehe Anatomie und Physiologie, und Klinische Medicin, Band., XXIV. 1862, p. 361.

§ De la Pathogénie des Hemorrhagies Cérébrales. Thèse, Paris, 1866, pp. 10, *et seq.*

tion, the exclusion failed to be so positive as regards the other antecedents, for the maternal grandmother had been subject to "a kind of fainting fits." Before leaving the subject of hereditary predisposition, let me point out Case No. 107 in table of female epileptics, the details of which will be given page 222. The woman, when quick with child in her first pregnancy, was seized with epilepsy, accompanied by mania, only recurring at every subsequent pregnancy. All her children but the first, are born epileptic, thus evincing the pernicious influence of the disease so strangely attacking the mother. Case No. 2, of the same table, also exhibits the transmission of epilepsy to the child, who died with fits, after birth. Finally, in Case No. 18, we find an intemperate mother with a daughter epileptic, whose two children died in fits.

If we turn from these instances, set down to hereditary morbid taint, to those in which marriage of consanguinity is assigned as the predisposing cause of epilepsy in offspring, the number, though too small to allow by itself any conclusion, increases, however, the very cogent evidence of more multiplied examples brought forward by Morel, Chazarain, Bemiss, Trousseau, and others. The two females on whom this influence was traceable, were each the issue of first cousins. The brother of one was idiotic, and she herself epileptic imbecile. The other, now twenty years old, and her youngest brother, four years of age, exhibited congenital hernia, which, according to Morel, is an infirmity not seldom visiting upon children by

marriages of consanguinity. This second girl, of sound mind, troubled with nocturnal epilepsy since childhood. Her next younger sister, free from any infirmity, has, however, an idiotic type, with flattened skull, small forehead, large ears, body of small size for her age, and is weak and nervous, the establishment of menstruation having been delayed beyond the age of sixteen. The little brother was, over a year ago, seized with paralysis of the right leg, he had also oblique inguinal hernia on the right side, and as this had become painful, and difficult to maintain reduced by a truss, assisted by Dr. L. B. Edwards, I performed Wood's operation, using a silver wire instead of thread. When I last saw the child, seven months after the operation, the paralysis and the hernia had not reappeared. I could add another case, not included among those here compared, and bearing a very significative proof of the evil consequences unalienably fixed upon children by marriages of consanguinity. The patient, a young West Indian, from Trinidad, belongs to a family mainly perpetuated by marriages between cousins, as I learnt from his mother, whose grandfather — himself issue of first cousins — died insane. He was married to a second cousin, and had by her a son paralytic, a daughter with chorea, and two more sons perfectly healthy. These two latter married respectively, a second and a first cousin; the one had three children, and of them, one female epileptic; the other had two daughters, one monomaniac and another, the mother of this patient. This lady

married a cousin, who died with yellow fever, leaving one only child, who is the young man in question, now 22 years old, and who became epileptic at the age of sixteen, after having had attacks of absence since childhood. Three years ago, the mother took him to Paris, to consult Trousseau, who was the first to call her attention to the hereditary influence rendering all hopes of recovery extremely doubtful. Deeply impressed with such an opinion, and upon closer investigation into the facts she communicated to Trousseau, this lady has been able to count 128 of her antecedents, most of them issue of the same blood, and among whom there were: five insane, seven paralytic, four epileptic, two with apoplexy, two with chorea, and finally one who had committed suicide. The subject is of such a cardinal importance, that I cannot refrain from strengthening my own evidence with that of Trousseau: "I once attended," says he, "the family of a Neapolitan gentleman, who had married his niece. There was no hereditary taint, and yet of his four children, the eldest, a girl, was very eccentric; the second, a boy, was epileptic; the third was of perfectly sound mind; whilst the fourth was epileptic and an idiot. A friend of mine who also married his niece, had four children, one of whom was seized at birth with grave convulsions, and another son is epileptic and an idiot. Not long ago, I saw with Messrs. Moynier, an epileptic boy, the son of first cousins; and shortly afterwards I had the opportunity of observing two analogous cases; one, that of a young man, aged 32; the other,

that of an idiotic child subject to epilepsy. Now that I carefully inquire into the question of consanguinity, whenever I see deaf and dumb individuals, idiots, and epileptics, I can scarcely tell you how great a share this influence seems to me, to possess in the causation of these affections." *

There are, as noted by the learned translator of Trousseau, prominent authors who do not attach this importance to marriages of consanguinity, thus maintaining their innocuousness. Lest I should weary the reader by a lengthened enumeration, I will only mention Robinson, Howe, Elliotson, Mitchell, Bemiss, Bergman, Erlenmeyer, Liebrich, Devay, Boudin, Morel, Chazarain, Trousseau, and then state that the evidence they have accumulated is so ponderous as to make it wholly irrefragable. Assuredly, such examples as those presented by A. Voisin,† deserve consideration, but to these individual facts, I would repeat with Trousseau: it has never been said that unions between near relatives were necessarily and fatally followed by evil consequences. And, it may be yet, added, as properly suggested by E. Crossman, that probably injurious results do not attend, where the uniting parties are not impressed with the same hereditary imperfections, in consequence of a previous cross having neutralized the tendency.‡

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* Op. cit., p. 86.

† Comptes Rendus de l'Académie des Sciences. Paris, 1865.

‡ British Med. Journal, April 13, 1861, p. 401.

The influence of consanguinity, is, perhaps, the most entangled subject of pathogeny, and to disengage its thread, is a difficult task to accomplish. However, to be consistent with the facts observed, we have to acknowledge, at least, as very probable, the injurious influence of consanguineous marriages, a conclusion arrived at by Falret, after a critical consideration of the evidence bearing on the question.

It is evident, by referring to the table of male and female epileptics, that the outbreak of the disease occurred before the age of puberty among the majority of cases, in which there was traceable any hereditary taint. And it seems, therefore, plain, as already established by Romberg and Reynolds, that hereditary epilepsy is usually developed before puberty, and at an earlier age than non-hereditary.

CHAPTER IV.

ACCIDENTAL CAUSES.

The fact that epilepsy is, doubtless, a hereditary disease, makes us assume that a susceptibility, or diathesis, is transmitted from parent to offspring; but, as we are deprived of means to distingush this diathesis, so are we equally without guide as to when the disease is shorn of these essential characters. Hence it is difficult to estimate always in a positive manner the influence of accidental causes in the development of epilepsy. The paroxysm may come on in a subject, where the hereditary taint, though existing, can not yet be traced as the primary cause of the disease, the outbreak of the spasmodic phenomena being apparently induced by some occasional circumstance suddenly arousing the unsuspected predisposition of the medulla oblongata, just as it would have happened under conditions unlike the above inherited one. Yet, there are instances in which the original influence of accidental causes is too evident to permit us doubt of it, and to the difficulty already alluded to is due that hereditary predisposition has been altogether disregarded by some authors, whilst others, on the contrary, attach to it the greatest importance in the etiology of epilepsy.

Here would be the place to allude to the distinction between centric and eccentric epilepsy, but there is no

necessity for me to expatiate on the subject. Every one knows that the first of these terms applies to those cases of epilepsy cured on removal of the peripheral exciting cause, whereas the latter cases, conversely, recognize their source in a disturbance immediately deranging the brain. It would be inferred from the preceding remarks on the pathogeny of epilepsy, that, properly, there is no more than one form of the disease, acknowledging its seat in the medulla oblongata. A centric epilepsy, *i. e.:* one originating from the centre, without previously bringing into action the incident portion of the diastaltic arch necessary for reflex movements, is a distinction neither accurate in point of fact, nor up to the present notions of physiology. Epileptic attacks — like every direct or reflex action of the spinal system — are *always* excited. This is a truth proclaimed by Marshall Hall. Spasms are not merely the expression of a force increasing without intermission until the movements reach their greatest intensity and inordinate character; they are, indeed, an ever-increasing force, but morbidly originating in the centre and incapable of manifestation, unless required to act by incident excitations, which finally exhaust such source of power upon constant requisition. Then, the paroxysm ceasing, the exalted resistance of the centre more or less rapidly regenerates itself to bring about a renewal of spasms, when again called into action. Therefore, peripheral lesions of the nervous system may be present without superinducing epilepsy, as long as they do not exalt the

action of the ganglionic cells in the medulla, and equally may the central lesion exist and spasms should not follow, unless incited by some peripheral irritation. In any case, the occurrence of such strange phenomena, familiar to any one inquiring into the pathology of epilepsy, has been verified by the experiments of Brown Séquard, showing that; when epilepsy appears in animals after injury to the spinal cord, and, therefore, acknowledges its source in the nervous centres, the fits have a peripheral origin. A very striking evidence of this seeming anomaly I saw in a case shown to me, at Bellevue Hospital, by Dr. Stephen Smith. An extensive surface of the parietal region had been removed by Dr. Smith in consequence of traumatic injury of the skull, attended with epilepsy, and the patient, a man, distinctly stated that the fits were always preceded by a cramp starting from the fingers of the right hand, corresponding to the injury sustained by the brain. Moreover, to ratify that the fits are induced by peripheral irritations we find, that they are usually arrested if the *aura*, or peripheral irritation, be prevented from reaching the nervous centres. I could not overlook one of the most undeniable evidences of this assertion, due to Odier and which I borrow from Portal.* A soldier, says Odier, had received a sabre-cut in the head, of which he thought himself cured, when he commenced to be seized with frequent cramps in the

* Op. cit., p. 212, and Odier. Médecine Pratique, p. 181.

little finger of the right hand. These cramps continued a few months, in spite of remedies prescribed by Odier, thereupon increased in frequency and intensity, reaching the wrist, the shoulder, and finally the head, and always ascending as an *epileptic aura.* Indeed, they ever were precursory of epileptic attacks.

To arrest such cramps, Odier advised compression of the nerves, by means of a string so disposed, " that by tightening one loose end between the vest and shirt, the arm was strongly tied around at two places, between the shoulder and the elbow, and between this latter and the wrist, thus completely arresting the pulse in this side," and with this ligature, the patient succeeded repeatedly in preventing the fits during three years. The patient in this way enjoyed the comfort of going everywhere without dreading an attack. The ligature became so necessary to him that he kept it constantly ready, even during the night, when many times aroused from sleep by the cramp of the little finger, he could at once prevent its dreadful consequences. Unfortunately, unmindful of the directions prescribed for his regimen, this patient gave himself to a debauch, superinducing indigestion and drunkenness, which prevented him resorting to the ligature to ward off an epileptic attack, from which he died. On post-mortem examination, the mark of the sabre-cut was found in the left parietal bone, a rugous protuberance, looking like caries, appeared, beneath the inner table of the bone, and under the dura-mater in this site, there was a sanguineous tumor, the

size of a large apple, soft, and filled with a great deal of limpid serosity." The connection of the peripheral irritation, with the production of the epileptic attacks, and their prolonged abeyance upon the arrest of the former, notwithstanding the persistence of the cerebral lesion, are too obvious to require further comment.

More instances might be cited in evidence of the peripheral excitation of the spasms in the so-called centric epilepsy, and we have already met with one of the kind in case VI. previously reported. Believing as I do, that the *aura* — meaning by it a cramp or other felt sensation — is in nowise a constant precursory symptom of the attacks, I, nevertheless, admit that, on the whole, everything warrants us in thinking that the paroxysm is always induced by peripheral irritations, generally unfelt, or not easily discoverable, and that even without removing the original lesion of the disease, the fits may be subdued as long as all source of disturbance be withdrawn from the nervous system. This fact is of the highest importance; and Marshall Hall declares it indispensable to frame any successful therapeutics of epilepsy. Brown Séquard has subsequently brought out many weighty arguments and experiments of value, to decide this fundamental question, which has no less chiefly attracted the attention of Sieveking, in one of the best reasoned, suggestive, and practical pages of his book on Epilepsy. It would be, therefore, presumptuous in me to insist in helping forward a truth so well established,

and upon which the limits of these inquiries forbid me to dwell at greater length.

I will avail myself of the classification adopted by Reynolds, to group the accidental causes of the cases here reviewed, into four categories: 1st, Psychical; 2d, Eccentric Irritations; 3d, General organic changes; 4th, Physical influences.

Nature of Cause.	Males.	Females.	Total.
I. *Psychical.*			
Mental work,	2	1	3
Mental disturbance,	2	1	3
Grief,	2	4	6
Fear,		1	1
Fright,		6	6
Disappointment,		2	2
Anger,	1		1
Anxiety,	1		1
Excitement,	1		1
	9	15	24
II. *Eccentric Irritations.*			
Dentition,	3	7	10
Indigesta,	2	1	3
Intestinal trouble,		1	1
Otorrhœa,		1	1
Disease of spine,		1	1
Uterine trouble,		2	2
Undeveloped uterus,		1	1
Onanism,	3	1	4
Venereal excess,	2		2
Rectal trouble,		3	3
	10	18	28
III. *General Organic Changes.*			
Over exertion,	2	4	6
Over exertion and intemperance,	1		1
Fatigue,	2		2
Fatigue and abuse of tobacco,	1		1
Intemperance,	18	7	25
Fever,	6		6
Brain fever,		1	1

Nature of Cause.	Males.	Females.	Total.
Chills,		1	1
Typhoid fever,	2	1	3
Yellow fever,	1		1
Small pox,		1	1
Scarlatina,	3	2	5
Rheumatism,	3	1	4
Diarrhœa,	2	1	3
Dysentery,	1	2	3
Pneumonia,		1	1
Syphilis,	4	2	6
Lead poisoning,	1		1
Excessive chewing and smoking,	1		1
Commencement of menstruation,		4	4
Arrest of menstruation,		13	13
Pregnancy,		1	1
Childbirth,		3	3
Puerperal hemorrhage,		1	1
Fright during childbirth,		1	1
Miscarriage from fright,		1	1
Fright during lactation,		1	1
	49	49	98
IV. *Physical Influences.*			
Blow on the head,	2	2	4
Fall on the head,	1		1
Fracture of the skull,		1	1
Fall and wound of the scalp,	1		1
Cerebral concussion,	1		1
Fall from a cart ; fracture of skull ?	1		1
Fall on the spine,		1	1
Blow on the back,	1		1
Insolation,	4		4
Attempted strangulation,	1		1
Fall on the arm,		1	1
Bite of a dog,		1	1
Ill treatment,		1	1
Severe punishment,	1	1	2
	13	8	21
V. *No accountable cause,*	49	66	115

General organic changes were apparently the most numerous accidental causes of epilepsy comprised in the foregoing table. Next in frequency came the eccentric irritation, comparatively surpassing in small proportion the psychical causes. I abstain from comparison as to the liability of the sexes to be influenced by the above causative conditions, because the larger number of female epileptics, prevents my drawing any correct conclusion on this interesting subject.

There are, however, among the preceding groups certain causes worthy of special reference and the foremost is intemperance. I have dwelt elsewhere on epilepsy, insanity, and other neuroses entailed upon offspring by intemperate parents, and from my experience of epilepsy I am also convinced that intemperance performs as great a part in effecting as in aggravating the epileptic paroxysm. The nervous derangements attending chronic alcoholism are: tremor, wakefulness, hallucinations, vertigo, sensory or motory paralysis, with epilepsy, dementia, or the acute symptoms of delirium tremens, mania and peri-encephalitis. However, not seldom epilepsy instead of outbreaking with the last is one of the earliest in the train of nervous disorders. Then, tremor, restlessness, indisposition to sleep and, above all, vertigo, precede the onset of the attacks, usually attended with maniacal excitement, delusion of sight or hearing, and a feeling of terror along with greater tremor of the limbs, and circulation considerably depressed,

with quick and irregular pulse. Hand in hand with these phenomena, the intellectual powers are impaired, dementia rapidly arising out of such condition. At this stage, the paroxysms with above symptoms may be often reenacted, but, less unfrequently, the spasms decrease, or even cease altogether, when the intellectual decline and paralytic symptoms reach their maximum. As a matter of fact it is generally believed that epilepsy is likely to manifest itself as the last accompaniment of chronic alcoholism with, or upon, attacks of delirium tremens and acute mania. It is also stated that, occasionally, *epilepsia potatorum* outbreaks upon total abstinence and that it often ceases if the intemperate desire be again gratified by the patient. Neither instance is here adverted to, and I have no design to discredit the last assertion, it being only question of epilepsy, preceded by vertigo, restlessness, trouble of the motory system, and as the unequivocal first consequence, without any other accompaniment, of the excessive use of alcoholics. Such instances have been more numerous in low women, though equally displayed by both sexes among the higher classes. Furthermore, the intemperate propensity in the latter cases has been on some occasions singled out as derived from parents addicted to excessive drinking, and even without their having been maniacal, demented, or conspicuously subject to the disorders begotten by intemperance. Morel, in the remarkable work previously quoted, especially refers to the overwhelming inclination to drink, ordi-

narily transmitted from an intemperate parent to offspring. This transmission may be readily traced out among the higher classes and, no less struck with its occurrence, Anstie observes: "I am inclined to believe that the great majority of the more inveterate and hopeless cases of alcoholic excess, among the higher classes, are produced by two factors, of which the least important is the circumstances of external momentary temptation, in which the patient has been placed, while the more momentous and weighty cause is derived from an inherited nervous weakness which renders all kinds of bodily and mental trouble specially hard to be borne. It need hardly be remarked that, in this view of the case, the fatal rapidity with which habits of intemperance exaggerate themselves is only what might be expected, seeing that the nutrition of the nervous centres would be still further impaired by each successive indulgence in poisonous doses of alcohol, and the power of moral resistance to feelings of depression and misery would be proportionately weakened."* So far as I am acquainted with the agency of intemperance, upon careful inquiry extending through a wide range of nervous diseases and great diversities of life, the instances fully confirm the just quoted testimony from one of the highest authorities on the subject. On the whole, then, I deem that the greater prevalence of nervous diseases now observed, acknow-

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* Alcoholism, in a System of Medicine, edited by Russell Reynolds. London, 1868, vol. II, p. 70.

ledges among its primary agencies the wide spread abuse of alcoholics, being no less staggered at the number of epileptics deriving their dreadful malady from this ruling habit, which, among the lower classes, adds itself to aggravate paroxysms superinduced by other causes. Although in chronic alcoholism the structural change undergone by the nervous centres clears the way to an unfavorable result, in early cases however, we may succeed in preventing it, and in the chapter on treatment, I will exemplify instances of epilepsia potatorum, in which the fits evidently were brought on to disappear upon the treatment instituted.

The commencement and the arrest of menstruation range conspicuously among the organic changes inducing epilepsy. The intervention of these causes appears more efficient than any other acting on the female sex. It is a trite remark that once fairly established epilepsy produces irregularities of the catamenia. I find in looking over the period of this complication in individual cases that, the younger the female the earlier the trouble existed, it being seldom delayed beyond the fourth month of the active progress of the spasms; whereas in those females affected after puberty, or at more advanced age, the mischief was not so particularly noticeable and obvious only in the severer cases. In those cases already reckoned in the first place, the cause from its inception showed no influence on the regularity of the menses, subsequently deranged. On the other hand, I fail to trace

any noxious association, or more frequent development, of the epileptic attacks with the age of menopausis. It would not be practically useful to lay before the reader a numerical estimate of the operation of epilepsy on the catamenia in the cases here compared. It is a very striking fact, however, that in all the forms of epilepsy dating from infancy, the establishment of menstruation was more or less delayed, the paroxysms having usually increased in severity on or about the menstrual period; and in one instance menstruation never took place. These results corroborate those of Georget, Beau,* and Marrotte.†

The subject, however, to which I wish chiefly to relate, is the commencement of menstruation, or its arrest, as immediate cause of epilepsy. On inquiring into the state of this function, I have excluded it from the etiology of paroxysms of unaccountable origin, although they either regularly occurred, or were severer during the defective catamenia. Neither have I set down the establishment of menstruation as cause of epilepsy, when any hereditary predisposition existed and was helped in its effects by the change of puberty. I have only registered instances in which, without hereditary taint or constitutional morbid derangement, the source of epilepsy did clearly lie in the disturbance to the natural onset, or to the regular return of menstruation.

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*Arch. Gén. de Méd. Tome XI, 2ème Série, 1826, p. 349.

† Revue Médico-Chirurgicale de Paris. Tome X, 1851, pp. 257, and 351.

There is one circumstance connected with the establishment of puberty which forms the dominant feature of this age, namely, the sexual orgasm. This secretely brooded fever is as peculiar to the human as to other animal species. When undisturbed in its operation, our organic frame assumes its procreative power without morbid shaking, but if held in restraint by constitutional or surrounding influences of any other nature, then all kinds of nervous disorders hang over the path marked out to the youth, who is soon reduced by the struggle to feebleness and depression. These facts, indeed, display themselves with more vivid and distressing forms in woman, condemned by social life to a position which draws after it her greater weakness to stand the sexual instinct. Maudsley accurately describes these feelings, as no less fertile source of insanity in woman. "The sexual passion — says he — is one of the strongest passions in nature, and as soon as it comes into activity, it declares its influence on every pulse of the organic life, revolutionizing the entire nature, conscious and unconscious; when, therefore, the means of its gratification entirely fail, and when there is no vicarious outlet for its energy, the whole system feels the effects, and exhibits them in restlessness and irritability, in a morbid self-feeling taking a variety of forms, and in an act of self-abuse, which on the first occasion, may, I believe, be a sort of instinctive frenzy, of the aim of which there is only the vaguest and most dim notion."* It is not,

* The Physiology and Pathology of Mind, 1867, p. 202.

therefore, difficult to understand how unnatural attempts to menstruation — first evidence of puberty — may superinduce the actual appearances of epilepsy in a constitution so much disturbed and irritable. And here, let me further remark, in a cursory manner, that, to this overlooked condition of distress, Anstie ascribes the chronic temptation to drink, and its secret practice, occasionally observed among females of the high class.* The form of epilepsy in question occurs, therefore, not in strong, but in debilitated young females, and the intensity and frequency of the fits increase in proportion to the depressed condition and irritability of the patient. There even appears in many cases, a marked coincidence between the exacerbation of the epileptic attacks, and the state of the uterine functions, for the former subside as soon as the sexual organs — and with them the system generally — are relieved from unhealthy excitement by the established uterine discharge. Indeed, so far as I have had opportunities of judging, this fact in great measure explains, why, of all the varieties, uterine epilepsy seems the less baffling and easier amenable to satisfactory treatment. Yet, if the regular return of menstruation may bring about such happy result, it is no less certainly true, especially when epilepsy has been favored in its development by hindrance to the natural establishment of menstruation, that this may appear without, however, remov-

* Op. cit., p. 69.

ing the epileptic paroxysms. There is, under these circumstances, a very important but not easily eliminated agent, and that is, the ovarian derangement which accompanies uterine disease generally, and perseveres as main source of trouble in these cases. We have not as yet altogether abandoned, in treating uterine disease, the tendency to neglect, or leave on the back ground, the condition of the ovaries, carrying our sight not much beyond the cervical canal exposed by the speculum. There is a growing reaction against the consequent surgical interference that has prevailed to this day in uterine therapeutics, and by which we have been led to own that distortion or displacement of the uterus are not by far the constant actual cause of amenorrhœa, dysmenorrhœa, and other nervous disorders exhibited by females. Generally, there is in their etiology, some unknown morbid state of the ovaries, which cannot be cured or relieved by means locally acting on the womb. Entertaining these views, after failure in accounting for the severe symptoms by the morbid appearance of the womb, and after unavailing local treatment, I have mainly directed the remedy to the ovarian trouble, recovery following more regularly, and thus indicating that, it is not the uterus but the ovaries that principally originate such obstinate symptoms. The correctness of this statement is ratified by uterine epilepsy. I will report, hereafter, instances of it where recovery occurred upon removal of the ovarian irritation, but I shall now present a most remarkable illustration of how

important and persistent the ovarian trouble may be in uterine epilepsy.

Case XVI. *Arrest of menstruation; imperfect development of the uterus; ovarian irritation. Metastatic pharyngitis and enlargement of the parotid, tonsil and sub-maxillary glands. Attacks of petit-mal in day time and nocturnal spasms.*

A young girl, aged nineteen, became epileptic when fifteen years old, upon suppression of the menstrual function at its onset. She had previously enjoyed good health; her parents were living, and no evidence of hereditary taint manifest in the family. The spasmodic attacks always occurring at night, commenced with the first signs of amenorrhœa, and subsequently repeated at the time of the month corresponding with the dates of the three or four menstrual discharges which she had exhibited. The disease progressing, the epileptic seizures increased considerably in frequency, preserving their nocturnal character, the girl being through the day scarcely without respite subject to fits of *petit mal*. She did not bite the tongue during the convulsions, these and the minor attacks being followed by coughing, and of most injurious influence to the mind, depriving her of memory until rendering her nearly idiotic. Having satisfied myself that the uterine trouble was the primary source of the disease, I consulted with my friend Dr. Thomas A. Emmet, on the case, the 27th of December, 1862. Upon examination with Sim's speculum, we found the uterus hardly larger than that of a child four or five years old, reduced mostly to the neck, and movable in every direction. There was scarcely any congestion of the canal, filled by a white thick mucus. The ovaries and rectum showed great tenderness on pressure. The vagina and external genitals were naturally developed, but the patient's figure was that of a girl of nine or ten years, and the whole body weakly framed. We determined to employ the uterine tent to invite the blood to the imperfectly grown uterus, and thus increase its nutrition. A sponge tent was inserted every fourth or fifth day, and kept in only eight or ten hours, to avoid inducing any great irritation of the womb. This plan suggested itself not only from the lack of growth of the uterus, but from the signs of ovarian irritation with unaccom-

plished attempts to menstruation. In addition, the girl was put under a course of bromide of potassium, and belladonna alternately combined with quinine and ergotine. The bromide was carried up to doses of forty and fifty grains, repeated three times a day, until producing drowsiness, with confusion of ideas and hallucinations of sight, which rapidly disappeared on the diminution of the salt. She also had for some weeks an induced electric current — the extra-current of Ruhmkorff's medical apparatus — applied to the spine at bed time; was directed to have a nutritious diet, strong coffee, cold bathing, and other means proper to invigorate her feeble general condition.

Menstruation reappeared in the course of seven weeks from the beginning of the treatment, and after not having regularly returned for nearly five years. On this plan the frequency of the epileptic spasms gradually diminished with the re-establishment of the catamenia, the general health improved, and she was for a year released from nocturnal spasms — the mind from the beginning regaining its former brightness. It was necessary to continue with the sponge tent for nearly four months, during the last part of this time being only employed at the approach of the menstrual period. The local treatment, beside renewing the suspended function of the uterus, determined a general development, the mammæ growing larger, and the girl assuming altogether the characteristics of puberty. Although the menses became regular and the mind clear, the girl still remained subject to ovarian and uterine irritation, and to slight rare attacks of *petit mal*, always severer on approach of the menstrual period, but no longer having any marked influence on her mind and very much resembling hysterical paroxysms.

To relieve the fits of *petit mal* a seton was put to the back of the neck. I also tried hypodermic injections of woorara, renewed every third day. The woorara I employed was kindly given to me by Prof. Dalton, and I was led to use it encouraged by the facts communicated by Thiercelin to the Academy of Sciences, of Paris, November 12, 1860. The solution contained one grain of woorara to ten drops of distilled water; two drops — a fifth of a grain — were at first injected, and this dose gradually increased up to half a grain. The injections were practiced on the neck; and on three occasions in the traject

of the seton, as many as eight drops being then administered. The effects observed were: redness, with increased heat, and numbness of the neck extending to the head; dilatation of the pupil, dimness of sight, with tingling of the ears and dizziness — irregularity and acceleration of the pulse, raising from 73 to 85. As the fits of *petit mal* persisted as frequent as ever, and as they even occurred twice a few moments after the injections, these were discontinued upon fourteen applications. I did not think of examining the urine, excepting that secreted the day following a nocturnal spasm, but then the presence of sugar, which seems to attend the injection of woorara, did not attract my attention in this sense as I had detected it before upon the above circumstances and outside of any employment of woorara. I need not say that, whilst this treatment was tried, the bromide and other internal remedies were discontinued.

I now return to the progress of the case. The spasms continued, during the year 1864, and the early part of 1865, to assail her in the night every six or seven months, preceded by exacerbation of the ovarian trouble, with great pain in the hypochondria. In the autumn of 1864, at the time of the catamenia, she was affected with painful enlargement of the parotid, tonsil, and sub-maxillary gland on the right side. I thought at first, this possibly due to the bromide of potassium, which she used then in thirty grain doses, three times daily; but soon I rejected this idea, since nothing similar was observable in the glands of the opposite side. Upon closer investigation, I found that the swelling commenced with the menses, and that the right ovary, exceedingly sensitive to abdominal pressure, was the seat of a severe pain, which the patient principally referred to the lower part of the back and loins. The glandular enlargement passed off with menstruation, and did not require any local treatment. Two months after, the same ovarian symptoms occurred during the catamenia, the girl being seized with tonsilitis. Finally, in January of 1865, the menses being retarded, she had a third attack of tonsilitis. It is true, that on this occasion the patient exposed herself to cold, which she supposed to be the cause of her complaint.

The above described course of treatment, with addition of counter-irritation, and galvanic electricity, applied to the ovaries, was kept up with benefit, but never sufficiently to eradicate the source

of trouble. The ovarian irritation rested ever ready to recur, with tenderness and congestion, at times increasing the size of the ovaries so much as to make them appear affected with a tumor. Laboring under these alternatives, the girl went to Europe in the summer of 1865. Previous to her departure, I saw her in consultation with my friend Professor Van Buren, who thought that the change of climate and life would prove beneficial to complete what such a persevering treatment had failed to achieve. I advised the patient's mother to see in Europe Prof. Scanzoni and Drs. Sieveking and J. W. Ogle. Having gone to Paris and discontinuing there my directions, the *petit mal* and nocturnal spasms recurred with their former severity, breaking down the general health of the girl. In this depressed state she was taken over to London, to consult Drs. Sieveking, Ogle, and Mr. Graily Hewitt. They directed the course of treatment previously pursued by the girl, insisting on the necessity of a more active life, which I had unsuccessfully urged all the time to the girl's parents. They further advised, as already practiced here by myself, to avoid local interference with the uterus, and mainly rely on stimulants and tonics, with general means, to remove the ovarian irritation. The girl returned from England in December of 1865, much improved, and replaced herself under my care until the spring of 1866, when she ceased to see me. She was then subject to very slight attacks of *petit mal*, and to the obstinate ovarian trouble, less frequently attended by nocturnal spasms. The intellectual faculties were unimpaired, and the general health better than before.

On considering the relation of symptoms, and the effect of treatment employed in this case, I have no doubt that the epileptic attacks were consequent and principally dependent upon the ovarian derangement. To the difficulty in completely mastering this latter, rather than to any other cause, I attribute the alternatives puzzling the treatment. The metastatic sympathy between the ovaries and the pharynx I have likewise discovered in other instances, forming

the subject of a paper read before the New York Obstetrical Society, April, 4, 1865.*

The hypodermic injections of woorara, which for the first time I tried in this case, I have further employed in four more, of which reference will be made in the chapter on treatment. I will, however, anticipate that my experience, though indeed too limited, has not been as satisfactory as that of Thiercelin, who saw the severity and frequency of the fits directly controlled by the endermic use of woorara, along with valerian and other means. Neither are more decisive than mine, the results of the interesting researches lately published by Voisin and Louville.† They have recorded, beside the phenomena above noticed, ptosis, diplopia, drowsiness, and sugar in the urine, among the ordinary sequelæ of the administration of woorara. The experiments were undertaken in twelve epileptics, over fifteen years of age, and as already stated, the above authors did not see that the substance was noxious, although they could neither praise its efficacy in epilepsy. The treatment was commenced with doses of two-tenths of a milligram, and gradually increased until reaching 18 centigrammes for hypodermic injections, 38 centigrammes for endermic application (blisters), and 40 centigrammes when administered by the mouth or rectum.

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*See New York Medical Journal, July, 1865. An abstract of the above case was reported in this paper.

†Journal de l'Anatomie et de la Physiologie. Paris, 1867, pp. 113, et seq.

No less important than the causative agencies already considered, and still among the group of general organic changes, are those relating to childbirth and the puerperal state. Instances of this kind are not of infrequent occurrence. More rarely, however, pregnancy becomes the inciting cause of epilepsy, and the example which fell under my observation, at the Hospital for Epileptics, was attended with too many interesting phenomena to pass unrecorded in detail.

CASE XVII. *Pregnancy. Epilepsy and mania. Hereditary transmission of the disease to offspring.*

Rosanna P. . . . . married, aged thirty-five, born in Philadelphia, admitted May 6th, 1867, into the Hospital for Epileptics. Has been a housekeeper, of temperate habits, and neither of her parents had epilepsy or any other nervous affection. She was seized with epileptic convulsions in 1847, when quick with child in her first pregnancy and gave birth to a fine boy, who has kept in good health to this date. Convulsive paroxysms recurred at every subsequent pregnancy, and she is quite positive that never at any other time, nor during labor, although convulsion along with maniacal excitement have supervened immediately after childbirth. She has six children, all but the first epileptic, and is now four months advanced in pregnancy. The fits usually appear at night, once or twice a week, since the very beginning of gestation. She awakes from her sleep, feeling a strange aura all over her, with glottic spasms, but generally has no time to speak before the convulsions come on, and completely losing consciousness, froths at the mouth and bites the tongue during the paroxysm. Her mind is a good deal impaired, she is dull, and the convulsions alternate with fits of mania, which render her taciturn, and inclined to commit suicide. Speech and hearing unimpaired. Sight dim since last February. Opthalmoscopic examination showed: both optic nerves swollen, with outlines a little indistinct, but otherwise nearly normal. Blood vessels full. Vague haziness of the left retina. Iris of a deep brown.

Tactile sensibility considerably diminished in the right arm and hand with tingling sensation in the fingers. Negative signs of paralysis in the limbs which feel constantly cold, their temperature ranging from 94° to 96° Fahrenheit. Skin dry; perspiration deficient. Pulse 64, slow and feeble, raising in force and up to 88 during the paroxysms. Respiratory organs healthy, no cardiac murmurs. Appetite poor; obstinate constipation. Urine acid, specific gravity 1030. No evidence of albumen or sugar. Urates and triple phosphates abundant. Upon speculum examination no abnormal state or ulceration of the womb was detected, although the patient complains of having occasionally had copious flooding during menstruation.

She was kept in observation, and it is useless to dilate on the treatment which offered no particularly remarkable effects. The bromide of potassium was given in large doses — forty grains with tincture of rhubarb, and alkaline tepid baths, nutritious diet, exercise, etc., were also prescribed. The change in her state seemed not very striking up to September 1st, she having had a severe turn of convulsions and mania in June, and another in August. I have been unable to learn the further course of this case, the report of which is abstracted from notes by Drs. Edwards and McClung, Assistant Physicians to the Hospital.

The influence of gestation on the development of epilepsy has been noticed by Van Swieten, Fernel, Schenkius, Lamotte, Tissot, Landré-Beauvais, Maisonneuve, Herpin, Sieveking and others. In the majority of their examples, however, the preexisting spasms were either augmented or, more frequently, arrested by gestation. Consequently, the foregoing is a more rare occurrence of epilepsy, evidently, superinduced by pregnancy. Sieveking states,* without comments, that: one of his patients became epileptic during

* Op. cit., p. 135.

pregnancy. Van Swieten speaks of a woman, subject to epileptic seizures every time she was pregnant with boys, and Lamotte * records a similar curious phenomenon: a woman, who was pregnant eight times, had epileptic fits on every occasion that she conceived a male child, the paroxysms never appearing upon conception of a female child. In my own patient the sex of the fœtus had no special influence in the production of the fits, but one not less primary feature of the case is the hereditary transmission of the epileptic taint from mother to children. Brown Séquard † has shown that guinea-pigs rendered epileptic by lesion of the spinal cord, usually entail the convulsive disease upon offspring. In this example, it should seem as though the exciting cause, after impressing the mother in her first pregnancy, without reaching the fœtus, left her subject to the same causative influence at every subsequent pregnancy, and beside capable of transmitting her predisposition to every child.

Though pregnancy, as just exemplified, has by itself proved capable of originating epilepsy, it is nevertheless a fact that marriage has been advised, principally to female epileptics, as a remedy for their obstinate disease. Herpin denies the beneficial influence of marriage on epilepsy, and quotes three cases respectively from Lanzoni, Cumming and Stegmann,‡ to manifest

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* Traité de Chirurgie. Tome II, p. 422.

† Proceedings of the Royal Society of London. Vol. X, p. 297.

‡ Du Pronostic et Traitement de l' Epilepsie. Paris, 1852, p. 520.

the worthless nature of their evidence. There are, however, a few well marked exceptional instances where the facts leave no room for doubt. Hoffman* describes a case of uterine epilepsy, in which marriage reestablished the regularity of the menses, with disparition of the epileptic paroxysms and other trouble. He further relates that: a woman whose parents had melancholia and who suddenly lost her husband, at the time of her menses, experienced a suppression of the catamenia by the shock, thereupon remaining with spasmodic paroxysms and great mental agitation at the menstrual periods. After long unsuccessful treatment a second marriage completely cured her. Prichard cites the case of a girl, for four years subject to epileptic attacks frequently, and always, returning about the menstrual period. She menstruated freely, and no other function seemed deviated from the natural state. The fits continued to recur until she married and became pregnant, when they ceased altogether. Based on this and other observations, including the above from Hoffman, Prichard is disposed to believe that: even if pregnancy should not take place, marriage may remove disorders connected with defects of the catamenia,† Ménard de Lunel reports that: "a lady of nervous temperament afflicted with epileptic fits, lost her husband at the age of 25. The paroxysms soon increased in frequency, attended

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* Opera Omnia. Tome III, p. 21.

† Op. cit., 190.

at the intervening period by nervous accidents, convulsive tremor, and great venereal excitement. This patient became secretly enceinte, and found herself at once relieved from all nervous derangement, growing fat and healthy after childbirth."* Sieveking† asserts that: "the mother of an epileptic lad for whom I was consulted was herself epileptic before marriage and continued so till the birth of a daughter, when the fits ceased and continued in abeyance for fifteen years, up to the time of my seeing her. It is true, the disease was transferred to the children in this case and may so far be an argument against the marriage of epileptics." The same author, while alluding to the influence of marriage on epilepsy, refers to "a widow of thirty-eight, who had fits from time immemorial, but had been freer from them since marriage and childbirth than formerly."

A lady, cousin of a female epileptic under my care, was seized with nocturnal spasms at the age of puberty, and continued so until she married, when the paroxysms ceased and have not recurred since for a period of seven years. This lady, however, has had four children, of whom, the first died with meningitis, and the third is troubled with infantile paraplegia. No hereditary taint of nervous disease is known to exist on the father's side.

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* Marcé. De la Folie des Femmes Enceintes. Paris, 1859, p. 119, quoted from Journal de Méd. et de Chir. Pratiques, 1834.

† Op. cit., p. 140.

This is not the proper place to enter into the physical disturbance that might be induced by continence — a subject closely allied to that presently discussed. That continence may become so injurious as to constitute a state favorable to the development of epilepsy, especially in women, is a fact which I doubt not. Though instances of the kind rarely present themselves in men, I have, however, met with cases in which the fits were directly connected with prolonged continence. A gentleman, 38 years old, whose nocturnal epileptic attacks, attended by voluptuous dreams and seminal emissions, resulted from his restraint of sexual intercourse, failing to be relieved by medicines and rules of diet, broke the severe habits and gratified the sexual appetite causing the arrest of his disease. Notwithstanding what is here stated and such exceptions as that brought out by Sieveking* to illustrate his views on the qualified veto that he would put upon the marriage of epileptics, I should feel very reluctant to advise the marriage of confirmed epileptics, no matter how mild the disease should be, and in females, even if it should proceed from manifest uterine origin. My meaning is this: we must not be emboldened by examples of fits associated to continence, nor by rare instances of their removal by marriage — not all clearly traceable to its operation; for should marriage prove effectual to arrest the fits it would not, however, prevent the transmission of epilepsy or some other

* Op. cit., p. 141.

neurosis from parent to children, as in the case cited by Sieveking, and in that I have above briefly alluded to.

As the counterpart of continence, I may now avert to venereal excess and onanism. The cases supplied from my own experience under the head of these eccentric causes of epilepsy are not numerous. It has been a received but gratuitous opinion that masturbation is among the chief sources of epilepsy. These views are condemned by later writers who strongly object to propound such causative relation between onanism and epilepsy. Whether the convulsive affection so frequently arises from masturbation alone, without any other agency, is, indeed, difficult matter to demonstrate; that the habit was the effect of the malady, has been a notorious fact in many of the cases under my observation. As original element of epilepsy, therefore, I believe that onanism and venereal excess have been in a great measure mistaken. Without intending to deprive either from any share in the etiology of epilepsy, I may say, from close clinical investigation, that they, at any rate, are more commonly the accompaniment of the epileptic disease. Not unfrequently, but by no means constantly, the sexual appetite is exaggerated in epileptics from the inception of their disease, and occasionally, such sexual hyperæsthasia is the forerunner of impotence when the trouble progresses. Whosoever has watched and attended to a great number of epileptics, will be able to bear testimony to their salacity or venereal orgasm. Neither is such derangement peculiar to epilepsy, for

it may likewise be observed among the incipient symptoms of other neuroses, and Trousseau has particularly pointed it out in ataxic patients. The early appearance of the trouble explains our perplexity between the chief part allowed by some to onanism in the etiology of epilepsy and the contrary views held by other authors. Hitherto, Leudet * is the writer who has insisted upon such erotic or salacious desires, exhibited by epileptics. Reynolds † disposes of the subject in this brief and rather indefinite manner: "In the male sex there is not unfrequently a morbid excitation of the genital organs, and this without it being evident that a causal relation exists between the two conditions. So far as I have been able to ascertain, it is as common for the epileptic to become salacious as for sexual excess to induce epilepsy. On the other hand I have met with instances in which venereal appetite has become extinct and this without any previous undue indulgence." Leudet, however, overlooked the true relation of the facts he so well describes, for he attributes to them a primary causative influence, disproved by attentive inquiry into the disease. Hence, onanism is put down by Leudet as determining cause in twelve out of sixty-seven cases of epilepsy that he studied. The psychical acts of the epileptic, their real nature and direction, are still very obscure. The motiveless and un-

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* Arch. Gén. de Médicine. Paris, 1843, 4e. Sérié, Tome II.

† Op. cit., p. 236.

controllable feelings, the strange derangement and delusions of the mental faculties, and mainly the rapidity with which the brain jumps from this morbid to a natural functioning, are, most assuredly, unaccountable operations hardly ever betrayed in their incipient form, when the epileptic, like the reasoning fool, conceals his morbid purports, which prevents us from ascertaining what so secretely undermines his moral nature. At the beginning, he may remain conscious and capable of partially controlling the manifestation of such morbid instincts; nay, anxious to discover a reason to his dreadful malady, in the absence of any other assignable cause, he may even deceive himself and the physician by acknowledging his salacity as source of the convulsive disease of which it is the exponent. I repeat it, such salacious appetite or venereal orgasm, which overmasters the patient, when mind and body are in no other apparent way disturbed, is usually the effect and not the cause of epilepsy. In saying this I have in view those patients only in whom onanism or venereal excess are predominant at the outbreak of the disease, which they greatly aggravate; patients, whose sunken eyes, without lustre, surrounded by a dusky or lead color circle, and tremulous moist hands, never naturally warm, and disturbed digestion, reveal in glaring manner the morbid instinct that rules them. I have so far met with four instances in which onanism was distinctly the cause of epilepsy, whereas not seldom I have myself witnessed the sudden outbreak of the salacious instincts,

when not informed of them by the relatives or the spontaneous avowal of the patient. The venereal orgasm bursts out at the intervals of the fits, but may as well precede or follow them, assuming then a true maniacal form. One of my patients, No. 45, table of male epileptics, suffered with spasms attended by maniacal excitement. He had never had sexual intercourse, nor was addicted to masturbation; yet, he would give evidence of satyriasis immediately before or after the fits, with terrible excitement, making him dangerous to persons near him. No sooner were the convulsions and maniacal attacks subdued, than the venereal desire also subsided. Recently, after passing a year free from attacks of mania, and five months without spasms, this young man had nine consecutive paroxysms accompanied by violent aphrodisiac symptoms, which yielded to packing in the wet sheet. Bromide of potassium given in drachm doses failed, in this as in former occasions, to determine any antaphrodisiac effects.

Another patient, No. 55, of the same table, with nocturnal epilepsy superinduced by mental overwork, pressed by my inquiry, wrote to me a history of his case, with the following about his feelings: "I must confess that your presumptions are correct. I practiced self-pollution from six to eight months after I was attacked with these fits: I never did it before. The average would be varying from once, to twice or thrice daily, or even with greater pertinacity and unsatisfied rage on the eve of a fit. I would not weary you with the

vain confession of prayers and resolves of one sinning, knowing the while how he sins, but, yet, finding himself led to the act without any intention or force to resist it. You may believe me or not; as for myself, I am unable to account for this venery that overcomes my whole being as anything but an evil result of my disease. If this avowal offers no other moral, it presents that of which you need not to be often reminded in the practice of your profession — how lightly we regard the blessings of health — and considering the self-abuse through which I pass, I think that I may say with the Psalmist: 'How fearfully and wonderfully are we made.'" Nothing remains to be added to this description, characteristic of an epileptic, and of the evil impulse and inability of the patient to carry out any course to repudiate it.

The most striking example of the venereal orgasm which may accompany epilepsy manifest itself in the subjoined instance:

CASE XVII. *Epilepsy. Paralysis of left arm. Sexual excitement.*

A girl, aged nineteen, slender and emaciated, was placed under my care on March 12th, 1865. From her mother I learned that she had the first epileptic fit at the age of sixteen; it came on suddenly in the morning, without any premonition beyond headache and great irritability the day previous. Never experienced any similar thing before, and though weak, enjoyed always good health. Neither was there any hereditary taint in her family. During the next six months the attacks repeated with increasing frequency, at first in the day time, but afterwards in the day and night. At this period, the power of voluntary movement in the left arm would be lost after a paroxysm to return slowly in a day or two, leaving behind a

good deal of numbness, with tingling in the fingers. After thus reappearing for a while, these symptoms became permanent, the arm remaining paralyzed. Menstruation, which had just set in seven months before the first attack, became scanty, attended with pain in the back and weakness in the legs.

Bromide of potassium had been unsuccessfully used for a year. When I first saw this girl, she was emaciated and pale, and had just recovered from a severe paroxysm. Her mind seemed in a lethargic condition, from which she would arouse occasionally, wandering. Forehead and neck with petechiæ. Pupils dilated, conjunctivæ hyperæmic. Tongue clean. Pulse feeble, 98, irregular. Respiration 18, at times sobbing. Extremities cold, with a purple hue. Heat of the rest of the body natural. Perspiration deficient. Left arm relaxed and powerless: not the slightest reflex contraction induced by pinching or application of heat to any part of the limb, and no evidence, however, of loss of sensibility of any kind. No tenderness on pressure, nor on passage of ice or of a sponge soaked in hot water along the spine. No trouble with the bladder. Bowels constipated. Urine scanty, high-colored, charged with urates. No albumen nor sugar; specific gravity 1025. My impression of the case was that some secondary cerebro-spinal congestion had been induced by the epileptic attack, probably giving rise to the paralysis of the arm and other symptoms. Ordered a large turpentine and assafœtida injection to move the bowels; a general tepid bath, with application of ice to the head, followed by a blister over the cervical region, and to take every three hours one pill with ext. conii gr. iij, and ergotine gr. ij. Diet, beef tea and milk punch. No material change made to this plan: two weeks passed and very slight improvement had occurred, the girl complaining of sleepless nights and of being excitable at bed-time. The mother then discovered, with painful surprise, that the girl had addicted herself to masturbation, and that she would openly do it day and night. This was a new phase of the case, and as the perverted feeling and restlessness did not abate, I resolved to administer forty grains of bromide of potassium every third hour. Four drachms and a half of bromide were exhibited in two days, until thoroughly bringing the girl under its influence: with low and fluttering pulse, often sighing or shivering; with eyes dull and flushed, and contracted pupils; very offensive breath and white coat of the tongue, thick articulation, and tottering gait, with inability to stand. Foremost among these phenomena was a heavy drowsiness, from which the girl would pass into a sort of catalepsy, or

often into a raving, accompanied with lascivious gestures, and words uttered in a thick and unintelligible manner. Such symptoms continued for nearly ten hours after suspending the bromide, and, curious enough, in the midst of delusions and other signs of the utmost effect of bromism, the girl had at 3 o'clock in the afternoon a severe fit attended by coma, lasting nearly four hours. She awoke from this attack extremely depressed, with quick and imperceptible pulse; action of the heart distinct, yet weak. The restlessness and nymphomania, the aberration of mind, soon returned with their former intensity, preceded by a fit. The bromide of potassium was withdrawn. She was directed to be packed in a wet sheet, with an ice-bag applied to the head, and to take a mixture with—

Acidi Hydrocyanici dil., ♏ v.
Tinct. Sumbul, ʒ j.
Aquæ dest. ad ʒ ss.—Misce.

The excitement gradually decreased, and two hours after being packed the girl relished an undisturbed sleep until 10 o'clock at night. At this hour the mixture was repeated, and she was enveloped in a freshly wet sheet. Calm sleep, though awaking at times, through the rest of the night. The next morning she is quiet, but excessively weak. Nourishing diet, with easily digested food, strong coffee, and stimulants. Packing renewed that morning and evening. Ordered to double the dose of conium and ergotine; to repeat the assafœtida and turpentine injection at bed-time, and to begin taking cod-liver oil after meals.

The improvement gradually increased; when the patient rallied and felt stronger, a seton was put to the back of the neck. The uterus was then examined, and found normal as to its position, without any ulceration or cervical discharge; yet, pain on pressure existed about the ovarian regions. The catamenia, although scanty, appeared regularly; but, during the four months after the attack above described, convulsions recurred frequently and severely a short time before or after the menstrual flow, attended with great distress and excitement, which would readily yield to packing in the wet sheet.

A nourishing diet, the continuance of cod-liver oil and large doses of conium with ergotine, in addition to the packing, or alkaline baths, gymnastics, and permanent counter-irritation to the neck, removed efficiently the nymphomania, and finally the epileptic attacks. No fits have repeated thereafter, and the girl, most closely watched by her mother, has exhibited no return of uncontrollable sexual desire, though ever remaining with a high hysterical temperament.

I may further allude to congenital phimosis, which in the case of males renders them especially prone to onanism. This malformation is not necessarily incompatible with health, though it may become a frequent source of troublesome local and general derangement. I am not aware of any writer having averted to the unmistakable influence which congenital phimosis has on the development of the respiratory organs, and which I remember having often heard particularly noticed by Professors Nélaton and Blache in their clinics, as far back as 1854. I have since had several opportunities of corroborating their statement, among other instances in that of a child, a patient of mine, with obstinate coughing, weakness, emaciation, arrested development of the chest and other appearances of phthisis, which were rapidly removed by the operation of circumcision. Nor has notice been taken of the complication of congenital phimosis and epilepsy, lately pointed out by Althaus,* who observed the malformation in eleven out of twenty-five consecutive male cases of epilepsy admitted at the London Infirmary for Epileptics and Paralytics. I have examined the genitals of almost every epileptic under my care, but I have not met with the congenital malformation in as high a rate as Althaus. Phimosis existed in sixteen out of the 130 male epileptics here reviewed; five of said cases are singled out of fifty-eight patients I have seen since I read

* The Lancet, February 16, 1867.

Althaus' interesting article, and their symptoms have been investigated more particularly in regard to the influence in question. There were on the first of August, 1869, seventeen male epileptics at the Hospital, and only three had congenital phimosis. The first of them was taken with fits of petit mal at one year; he is now twelve years old and is addicted to masturbation. The second patient, aged 21, was seized with spasms a year and a half ago, and is free from self-abuse. The third, aged 14, became epileptic at 9: he has an uncontrollable tendency to masturbation, and his penis and prepuce are considerably elongated. The operation of circumcision has been performed on him, with very slight benefit. I intend to operate also on the other two patients as soon as I become better acquainted with their symptoms, in order to appreciate better the influence of circumcision upon their epileptic attacks. I have, however, often made out, like Althaus, a conspicuous connection between congenital phimosis and headache, giddiness, noise in the ear, or other nervous derangement, and the above remarked trouble with the respiratory organs. As regards the sixteen epileptics with congenital phimosis I have observed, there have been only two in whom the malformation appeared instrumental in the causation of the fits. Reference has already been made to one of these cases, page 115. In this instance there was a clear sequence between the congenital phimosis, onanism, and the epileptic fits, inasmuch as the first paroxysm occurred imme-

diately upon masturbation, and the others commonly repeated every morning at the moment the urine, as it was voided, touched the constricted orifice of the prepuce. Circumcision, advised by Professor Van Buren and myself, did not immediately affect the fits, notwithstanding the removal of the degenerated nervous filaments in the cut-off portion of the prepuce. It is true that, as the fits did not cease directly upon the operation, the father stopped all further treatment of the patient, who became demented and was finally carried to a lunatic asylum.

It would prolong this chapter too much were I to give a detailed account of every causative condition of epilepsy grouped in the table page 207. The most important appear illustrated in the foregoing cases, and in those yet to be brought forward in connection with other subjects. Therefore, agencies will be now only exemplified which have more to do in exciting epilepsy than generally supposed, although they should not be considered as at all special, for their action may vary a good deal, according to the idiosyncrasies of every individual. The group of general organic changes comprises pathological conditions which are the expression of disorders without any close analogy to each other as regards their nature or their origin. However, every one of these morbid states entails upon the organism the depressed condition necessary to favor, through exciting circumstances, the onset of epilepsy. Yet, for the diseased action of the nervous system to occur, it is requisite that the blood should

undergo a modification inducing the nutritive change in the medulla oblongata proper to epilepsy. Consequently, whatever be the agency of each individual cause herein considered, exhaustion, or an impoverished or altered state of the blood, is usually the result. If this so disturbed blood renders not every individual liable to identical nervous derangement, it is, indeed, on account of the special circumstances of every case; and last, but most certain, of the idiosyncrasies which render each individual more prone to one than to another one neurosis.

I previously cited an instance where the mother, seized with frequent severe attacks of malarial fever, gave birth to a child "born with fits," and epileptic thereafter. In another patient, No. 99, Table of Female Epileptics, the first convulsive paroxysm occurred after an attack of ague, and returned subsequently along with fits of petit mal until inducing paralysis, with numbness and coldness of the lower extremities. Large doses of quinine, together with Fowler's Solution and the bromide of potassium, eradicated the spasms, while the fits of petit mal still continued, slowly decreasing in frequency and severity with the treatment, which has nearly lasted two years. The alliance between malaria and epilepsy has been acknowledged from the very early times of medicine. Hippocrates distinctly speaks in some of his aphorisms of the epileptic fevers—*febres comitiales*, and of their greater frequency during the spring and autumn. Modern authors have likewise related instances of

epilepsy directly following or disappearing after intermittent fever. Dr. Mackay, of Edinburgh, has recorded a very interesting case of malarious epilepsy cured by quinine. Handfield Jones, who has made important inquiries on the various disorders that malaria is capable of generating through the nervous system, on referring to the form of epilepsy now alluded to, cites cases observed in India by Lowe and Payne, in addition to that just mentioned of Dr. Mackay. Payne has seen "epileptic attacks ensuing as the result of malarious infection, which may have occurred long previously, and not have shown itself in paroxysms of ague at any period."*

I will simply add that I have met, not rarely, with epileptic spasms replaced by chills, with pale and cold skin supervening upon the premonitory symptoms of the former attacks. I am now treating a gentleman in whom the convulsive fits have been arrested for eight months, during which time he had occasionally experienced the precursory disturbance of his previous paroxysms, being actually reduced to a cold stage of rigors and uncontrollable shaking of the limbs for two or three minutes, followed by free perspiration of the head, with large red patches on the skin, lasting half an hour or longer. Thirty and forty grains of quinine, exhibited on repeated occasions throughout the day until affecting hearing, did not keep the attacks in abeyance. They have, however, diminished in fre-

* Handfield Jones—Clinical Observations on Functional Nervous Disorders. Phila., 1868, p. 139.

quency and severity since the patient has been taking daily, in divided doses, twenty minims of liquor potassæ arsenitis, and having injected hypodermically in the neck one-fiftieth of a grain of strychnine, in addition to cold shower-baths and a tonic regimen. It seems to me that under the foregoing circumstances the circulation of the medulla oblongata and brain proper has not been deeply affected, the trouble essentially involving the vaso-motor nerves of the arteries supplying the voluntary muscles and the integuments: hence the rigors and coldness of the skin. The irritation upon such arterial nerves cannot continue for any length of time without exhausting the vascular contraction and thereupon relaxing the blood-vessels, as evinced by the hyperæmia of the skin, with sweating and the rash already described. One of the male epileptics at the Hospital was seized in February of 1869 with intermittent fever of a tertian type, subsequently changing into epileptic fits beginning with vertigo, and repeating less and less periodically, until now occurring once or twice a month.

The connection between epilepsy and physical influences has frequently been shown. Besides the cases in this category already noticed, I will have occasion of citing some other examples of equal practical value. Now, however, a place must be conceded to that instance of fall on the spine attended by chorea and epilepsy.

CASE XIX. *Fall on the spine. Chorea and weakness of the lower limbs. Epileptic attacks.*

A girl, aged 10, was brought by her mother to consult me, June 6, 1865. Eight days before, she fell down stairs from a considerable height and struck her spine against the banisters. She was unable to raise herself, her legs were folded under her, and a feeling of numbness with tingling extended all over the lower limbs and back. In this powerless condition she was removed to bed, and then became very nervous, starting at the least touch or slight noise. She complained of pain across the head; but her mind remained perfectly clear. The physician, called to see her, ordered leeches to the lower part of the back, and a narcotic mixture. She awoke the next morning still complaining of pain in her head, and agitated by starts or a general twitching, more manifest in the lower extremities. These paroxysms at the end of five days occurred less frequently and in much milder form; but she experienced difficulty in walking, and particularly in ascending or descending the stairs. About this time and during the night she was seized with a violent epileptic paroxysm, in which for a minute the whole body rolled over and over, respiration became arrested, with frothing at the mouth, lividness of the face and neck, sweating, involuntary passage of urine, stertor, and coma, the girl not being therefore aware of the occurrence of such an attack. A similar one was repeated the subsequent night, and again the night before I saw her. When brought under my notice she presented the following symptoms: Pain in the head and neck; no alteration in the size of the pupils, which contracted readily to light. Feeling of constriction and tenderness around the abdomen. Pain on pressure along the spinous processes of the lower dorsal vertebræ, extending down to the sacrum, the same pain and difficulty being experienced on bending the body. Slight twitchings of the arms, with shrugging of the shoulders; legs and thighs benumbed with convulsive shakings. She could move and raise them; but when she was placed on her feet, they twitched and yielded under the weight of her body. Sensibility dull in the lower limbs, but unimpaired in the arms. Hands and feet cold. Bowels confined. She had perfect control of the rectum and bladder. Tongue slightly coated in the centre. Appetite good. Skin dry, with natural heat. Pulse 97, irregular, and firm. Respiration 24. Heart's action violent and irregular; no murmurs. Lungs natural, but respiratory sounds feeble. Tickling or pricking the feet excited jerking, and if re-

peated, complete stiffness of the legs. Urine acid, clear, and loaded with phosphates; specific gravity 1030.

She was ordered a blister to be applied to the lower dorsal region; to take two grains of extract of hemlock with equal quantity of ergotine three times daily; to use at bed-time a turpentine and assafœtida injection; to go for two or three seconds under the shower bath every morning, and, to be kept as much as possible in a recumbent position, and, as a matter of course, lying on her sides or stomach. It was also advised that a diet should be given consisting of easily-digestible food, claret wine, and coffee, and that she should at the same time avoid overloading the stomach.

The blister seemed to relieve her, as the shaking of the legs and the tenderness with constriction around the abdomen were mitigated. The state of the bowels also improved. She, however, had a fit four days after commencing the above treatment. The doses of ergotine and hemlock were doubled, counter-irritation was more actively kept up, with a red-hot iron occasionally applied to the dorsal region of the spine. From this time the epileptic fits ceased, and the shaking and trembling of the limbs, with pain of the back, and other symptoms, gradually passed away. She had continued free from fits for four months when I last visited her, and I afterwards learned from her mother that the cure had persisted until that date, August, 1868.

This is a typical case, clearly displaying the consecutive progress of the irritation from the dorsal region of the spinal cord up to the medulla oblongata, the convulsive paroxysms occurring precisely as in the experiments performed by Brown-Séquard, when injury to the lower regions of the cord was, after a period of about seven days, attended with epilepsy. Injury to the spine is not uncommonly acknowledged in the etiology of chorea; neither is it infrequent to meet with this latter supplanting epilepsy, or otherwise occurring in the intervening period of the fits. I have not seldom observed one convulsive disorder

merging on, as well as alternating with the other. The same has been remarked by Sieveking.* C. B. Radcliffe says: "I have frequently met with epileptic patients who were choreic at one period of their life, and the impression left on my mind from what I have seen is, that the chances of chorea being followed sooner or later by some other disorder of the nervous system are too much made light of." †

The bite of a dog, supposed to be rabid, was assigned as cause of the disease in case 39, Synoptic Table of Female Epileptics, but I doubted the fact, inasmuch as not the slightest hydrophobic symptoms ever existed before, or with any of the paroxysms. Nor is there any reason to believe that fright might have operated to induce the latter, on account of the long period of seven weeks intervening between the bite and the first convulsive paroxysm. The personal habits and peculiarities of the girl, on the contrary, indicated that the trouble emerged from a predisposition which, through some concealed inciting cause, gave rise to epilepsy and its attending maniacal excitement, attributed by the mother to the bite of a dog because this happened perchance a few weeks before the onset of the fits. The subsequent course of the case has confirmed the correctness of my views concerning the unknown origin of the disease. The maniacal element exhibits itself now very con-

* Op. cit., p. 29.

† Chorea, in a System of Medicine, by J. Russell Reynolds. Vol. II. London, 1868, p. 125.

spicuously, attended with cataleptic symptoms. After a series of fits which occurred on the 8th of September, she tore off with a hair-pin the gum from the whole alveolar process on the left superior maxillary bone, and pulled out one after the other every tooth implanted in it, *i. e.*, two incisors, the canine, two bicuspids, and the first and second molars, which were in healthy condition. She silently and most deliberately inflicted on herself such fearful injury without the slightest indication of pain, and would have persisted in tearing out the denuded bone if she had not been prevented by the strait-jacket. I ascertained that this analgesia coexisted with complete anæsthesia of the face and mouth, redness of the cheeks and ears, excessive dilatation of both pupils, and a fixed look of the eyes. The fits recurred accompanied by tonic spasms of the arms, and in the interparoxysmal period she remained in an ecstasic state, with total suspension of mental power and sensibility. This, indeed, is the most remarkable instance I have met with of anæsthesia attendant upon epilepsy. The hysterical element has evidently a special effect on the curious mental and cataleptic phenomena displayed by this patient, who has been presented to my clinical class at the University Medical College.

CHAPTER V.

FREQUENCY AND NATURE OF THE EPILEPTIC ATTACKS —AURA— PARALYTIC SYMPTOMS — APPEARANCE OF THE RETINA—STATE OF CIRCULATION AND RESPIRATION—CHANGES IN THE URINE.

The necessary limits of these inquiries prevent my examining the relations between the symptoms in the cases of the foregoing synoptic tables, especially, as may be seen at a glance, when the results do not essentially differ from, or conflict with, those of well-known authorities, and particularly of Russell Reynolds, who has given more prominence than any other writer to this important subject, in his valuable work on Epilepsy. I will not, therefore, discuss all these diverse symptoms, but will only enter into a detailed account of some phenomena to express my own view connected with questions which arise with regard to them and are still not distinctly established. The relative frequency of the paroxysms of petit mal and of those of general spasms, as well as of the two forms combined, and of the nocturnal attacks in the 306 cases here analyzed, was as follows:—

FORM OF ATTACKS.	MALES.		FEMALES.	
	Number.	Per cent.	Number.	Per cent.
Petit Mal	5	3.07	4	2.72
General spasms	110	84.61	135	71.01
Two forms combined	15	11.53	37	21.19
Nocturnal spasms	6	4.61	22	12.5

It therefore appears, as established by Reynolds,* "that the severer form of the attacks is by far the more frequent in both sexes." "Again, it is much more common for the severe attacks to exist without the slighter forms, than it is for the milder form to be present without the severe." "Further, it is extremely rare for the attacks of epilepsia mitior to occur alone." "There is no important difference to be observed in regard to sex, in relation to the two forms of attacks,"—excepting as to the attacks of petit mal (*epilepsia mitior*) and of the two forms combined, which, according to my estimate, are more frequent in females. The same greater frequency is further observed with nocturnal attacks in the female sex; and while these nocturnal attacks were uncomplicated in the males, there were among the females three with fits of petit mal, and a fourth with vertigo and headache in the day-time. Taking a wide view then of the subject, there is no remarkable dissimilarity between my ultimate results and those arrived at by Reynolds, although the respective percentages of our tables differ very much from each other, the proportion swelling considera-

* Op. cit., p. 138.

bly on my side. This may, perhaps, depend on the more numerous cases comprised in my calculation, whereas Reynolds's statistics are obviously too partial—twenty-nine males and twenty-five females,—to come nearest to the average ratio which cases bear to each other.

Hereditary taint appeared in :—

	MALES.	FEMALES.
Cases of petit mal	3	0
" general spasms	15	27
" two forms combined	4	6
" nocturnal spasms	1	3

The age of invasion for the attacks of petit mal was, in the five males, respectively: infancy, seven years, eighteen years, nineteen years, and twenty-six years; in the females: six years, fourteen years, fifteen years, unknown. It is no less curious that two more of the females with nocturnal spasms had father with cardiac disease, and became epileptic at the age of twelve. In another female, affected with petit mal and spasms at the age of seven, the mother had phthisis and the father cardiac disease. These results seem to confirm the opinion—that hereditary taint exerts a favorable influence on the development of *epilepsia gravior* rather than of *epilepsia mitior*, as also concluded by Reynolds; but they contradict his assertion—"That whereas in the male sex the absence of hereditary taint appears a predisposition to epilepsia mitior, such is not the

case in the female sex, the numbers in regard of the latter sex being equal. Where hereditary taint is present the tendency to escape attacks of *le petit mal* is equal for the two sexes; being for each as 2 : 1."* I do not believe I am warranted in concluding from the reduced number of cases here compared, that hereditary taint is without influence in the development of petit mal in the female sex. In regard to age and predisposition to petit mal, it appears that this form is more apt to invade between second infancy and adolescence, and the same holds good in reference to the two forms combined of the disease, excluding instances with unknown age of invasion of the attacks. These conclusions agree in every respect with those of Reynolds, who establishes: "that when the disease begins between the sixth and twentieth years, there is a stronger predisposition to the development of epilepsia mitior than there is either at an earlier or a later age; and also that when the disease commences during that period it is more likely to be displayed by both forms of attacks than by one only."†

Most assuredly the severer is by no means always preceded by the so-called milder form of epilepsy or petit mal; but I reject depriving this latter, as Reynolds does, of any direct agency in the development of the former. Exceptionally do we meet with long-standing attacks of uncomplicated petit mal, while the efficient part they take in the

* Op. cit., p. 141.

† Op. cit., p. 143.

production of general spasms, or genuine epilepsy, is not infrequently self-evident, and entitles them, therefore, to be looked upon as more than a mere coincident event. We know how vague an account epileptics usually furnish of the incipient stages of their malady, and their still greater propensity to pass by unnoticed the earliest vertigo, or other slight symptom of petit mal, fixing the invasion of the disease from the moment of the first spasms, which they endeavor to ascribe to some circumstance coinciding with their onset. And this is not a gratuitous assertion on behalf of what I advance, but a fact of every-day occurrence in the etiology of most every nervous disease. I can affirm that, with several of my patients, they, or their relatives, have stated that sudden faintness, giddiness, or vertigo, preceded for months or years the first convulsive paroxysm. One male, epileptic since infancy, had fits of petit mal during the first year. A female, also affected since infancy, had petit mal until 20 years of age, continuing thereafter with the two forms of the disease. In another, the milder form, beginning at 3 years, persisted until the birth of her first child at 18, to be then supplanted by general spasms, paralysis of the right limbs, incontinence of urine, and other symptoms. Finally, another female, again, has scarlatina at the age of 5, subsequently becomes deaf and for a short time insane, before exhibiting fits of petit mal, which last until 18 years of age, when they cease altogether on the supervention of general spasms. In all these instances the

petit mal is the initial stage, or paves the way for general spasms; whereas, it is one of the most common phenomena in cases of petit mal and spasms, to see the second disappear long before the first, which may persist and baffle the best conducted and most persevering treatment. Indeed, although the petit mal does not constantly supervene in the course of every case of epilepsy, yet I assume, upon personal observation, that it figures conspicuously among the premonitory symptoms of the severer form. And whilst I fully concur with Reynolds in believing that "the presence of *le petit mal* is, cæteris paribus, a sign of greater severity of the disease than its absence," I feel called upon to remark that the records of cases under my care do not indicate that "the petit mal is an additional phenomenon, present in those cases where the frequency of epilepsia gravior is at its highest." Nor—"that it appears to augment the frequency of the *haut mal*."* Of 38 patients (10 males and 28 females) where attacks of general spasms occurred with extreme frequency, in periods of recurrence from one day to one week, only one male and three females exhibited petit mal. Furthermore, with the male the petit mal ceased whenever the spasmodic paroxysms assumed their greatest frequency—from fifteen to eighteen fits in twenty-four hours—followed by maniacal excitement. One of the females had as many as one hundred and

* Op. cit., pp. 141-154.

eleven successive paroxysms in a day, and was also subject to great hysterical excitement and aphonia after the attacks. She never had been troubled with petit mal. In the three other females, with both kinds of fits combined, the convulsive paroxysms increased in frequency. without, however, augmenting the attacks of petit mal. One of them had the spasms only at night, and the more frequent the diurnal fits of petit mal the safer she remained from nocturnal attacks. The other female, with dysmenorrhœa, exhibited general spasms only during the catamenia while asleep, and she sometimes had as many as from 18 to 32 fits in one night. The petit mal appeared in the intervening days between the menstrual periods, when she would often complain of vertigo and absence, never attended, however, with convulsions. In the third female the first spasms set in immediately after an attack of ague, and soon became associated with petit mal, producing chilliness, with a feeling of horror and momentary loss of consciousness, followed by cold perspiration of the lower limbs. The convulsive fits, in the beginning nocturnal, lost afterwards this character and repeated frequently at all hours, thereupon inducing paraplegia. At this stage the petit mal stopped completely, but gradually returned as the spasms decreased in number and frequency.

I previously asserted that epileptic attacks are always excited, and consequently I look upon the aura as a most important element in their production—strictly meaning by aura the felt or unfelt incident

excitation requisite to the outbreak of the fit. This trouble of innervation may momentarily anticipate the epileptic paroxysm, or it may for minutes, hours, or longer time herald it, as in that patient who had foul breath for a day before the fits. In other instances of vertiginous epilepsy, the aura—as remarked by Trousseau—may alone constitute the attack. The difficulty which often exists in ascertaining the origin of the unfelt peripheral irritation is easily understood, especially when it may not always arise from the same site, and yet be capable of setting into action the morbid faculty of the medulla oblongata. All that needs to be established here as a fact of cardinal importance is, that peripheral excitations of any kind suffice to induce epileptic paroxysms; that these paroxysms do not exist unless there be such previous excitation; and, that everything tending to set it up efficiently tends to usher in the epileptic attacks. It is one of the greatest discoveries of Brown-Séquard to have proved by most skilful vivisections this peripheral origin of epileptic fits, even when their source proceeded from the nervous centres.

The aura is not infrequently distinctly perceived by the patient, who, thus admonished of the approaching attack, may ward it off by barring the progress of the mischievous incitation. Felt auras vary with the phenomena which characterize them, and pertain to the mental, sensorial, or motorial faculties, when not involving the vascular and secreting systems. The relative frequency of a perceived aura in the 306 cases

here considered has been: 13 times, or in 10 per cent. of the males; and 15 times, or in 11.53 per cent. of the females.

The precursory disturbances among males were:—
Hallucination of sight.
Tinnitus aurium.
Wild state of mind and cries.
Chills.
Perspiration in the head and arms.
Foul breath for a day before fit.
Vomiting.
Pain at the stomach.
Indescribable sensation at the stomach.
Glottic spasms.
Cramps in right leg.
Spasms in the arms.
Cramps in right hand.

Among females:—
Hallucination of hearing.
Vertigo and crying.
Vertigo and headache.
Chills and wandering of mind.
Smell of smoke, and numbness in left arm.
Bad taste and choking sensation.
Uncontrollable running.
Nausea.
Sickening sensation at the stomach.
Pain in the tongue.
Pain in the right side of the body.

Tingling of the whole body.

Cramps proceeding from left hand.

Cramps proceeding from little and ring fingers in right hand.

Tickling sensation in fingers of left hand.

It is curious that the aura and petit mal were present only in one female, aged 27, with attacks of vertigo and absence, and convulsions preceded by pain in the right side of the body. The petit mal commenced in infancy and continued alone until the age of 20. She was besides troubled with night-blindness. Ophthalmoscopic examination exposed in the right eye indistinct borders of the optic disc, vessels full, numerous minute spots of pigmentary retinitis. In the left eye, nerve very indistinct in outline, patches of exudation above and below it, and along the turgid retinal vessels. Spots of pigmentary retinitis at periphery of fundus. According to Liebrich,* idiotcy usually accompanies pigmentary retinitis, which is besides peculiar to offspring of consanguineous intermarriage. Neither circumstance was observed in this case, nor was the hereditary taint, admitted by Gräfe as constant, an etiological element of the above affection.

Another very interesting instance of señsorial aura, admonishing for prolonged time the approach of epilepsy, is that of a man 21 years old, troubled with tinnitus aurium and the following feelings I describe

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* Deutsche Klinik, Feb. 9, 1861.

with the patient's own words—"I can tell for an hour or two before going into one fit that it is coming on, by a peculiar lameness that comes over the legs, and then I have dreadful pain in them extending to the groins, hearing at the same time the ringing of distant bells, which recedes as the fit approaches. My feelings become so terrible that I wish for the fit to follow; but still I struggle against it for an hour or two, until the conflict is over." I have seen this patient in the just described condition, with an expression of bewilderment, struggling to overcome the attacks which he could never avoid, and which, however, were instantly put off by running under a cold shower bath. In this case intemperance induced the fits without any other previous trouble. The first attack took place in December, 1863, and since that date continued once, twice, or several times in the week, having paroxysms attended with the above terrible sensations, giddiness, general numbness, and an indescribable feeling as if he were going to die, with great horror and tingling sensations, sometimes in the fingers, and at others in the legs. Large doses of bromide of potassium—two scruples three times daily—and hypodermic injections of $\frac{1}{60}$ gr. of strychnia in the thighs, in addition to a tonic regimen, cold shower baths, and gymnastics, arrested the convulsive attacks in the course of four months.

In a female, aged 36, an odor of smoke and numbness in the left arm announced the fits, caused by fright during child-birth. The left arm also remained

benumbed for three or four hours after the spasms. The absence of cardiac disease discountenanced here the idea of cerebral embolism, put forward by Hughlins Jackson* to explain loss of smell and aphasia attendant with epilepsy. Yet, the condition of child-birth, during which the first epileptic seizure took place, is favorable to assume that some migratory clot detached from the uterine blood-vessels might have been carried out to plug the cerebral arteries. In this instance there was no variation in the size of the pupils, nor impairment of sight, both eyes looking normal to ophthalmoscopic examination. No symptoms of motor paralysis accompanied the diminished tactile sensibility in the left arm. The sense of smell was totally lost on either side; and on no occasion did aphasia supervene upon the fits, the patient having been closely watched at the hospital. Cases similar to this have been reported by Delasiauve, Ransom, and other authors, quoted by Jackson in the interesting article already cited.

I could not pass unnoticed the case of a girl, aged 22, with general spasms, always occurring at day-time, preceded by an uncontrollable impulse to run; perfectly conscious of everything around her until she would fall down in convulsions. If held at the start and violently shaken, the paroxysms would occasionally stop, but their increasing frequency obscured her memory and deranged her mental faculties. I could

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* Med. Times and Gazette, April 30, and August 13, 1864.

not ascertain the exact age of invasion of the disease; but the patient's uncle, with whom she was living, thinks that at about the age of 12 she was afflicted with "running fits," the intellectual faculties having gradually failed throughout the last two years. The paroxysms usually occurred in the forenoon. I ordered that the girl should at different times try violent exercise with a skipping-rope; a seton was put to the back of the neck, and bromide of potassium and conium were besides administered in full doses. The perturbation caused by the violent jumping evidently proved of great avail; no fits repeated in the course of three weeks after commencing the above treatment, joined to a tonic regimen. At this time the girl experienced a very severe attack, and another at the end of five weeks. The intellectual faculties regained brightness and improvement persisted, when in March, 1867, she was taken with measles, and from that moment to December, 1868, no fits of any kind have recurred, although the girl exhibits a highly hysterical temperament. Violent exercise in a trapeze controls the spasms in another patient of mine, who of his own accord resorted to this practice, believing that bodily fatigue would keep off the convulsions. This young man, sent to me by my friend Professor T. G. Thomas, had to suffer amputation of the right arm upon a railroad injury, and the operation had no effect whatever on the epileptic fits. "In a case of spasms of some muscles of the jaw and face, preceded by a sensation of pricking on the cheek, and occurring several times

a day, in a boy seven years old, Brown-Séquard found that violent exercise on a swing always prevented the fits when the patient had time to run to the swing before the muscular contraction had begun. By that means, which never failed, the boy was many hundred times saved from his attack before being completely cured of it, which occurred on the coming out of a molar tooth, which, however, had not given the least pain."* I could not explain what prompted me to try the plan of violent exercise under circumstances so distinct from those of the boy treated by Brown-Séquard, and by whose example I was guided: but the plan did certainly benefit the girl under my care. In regard to the disappearance of epilepsy upon the outbreak of an eruptive fever, the fact has been often noticed, and I have further seen obstinate daily fits of petit mal and spasms in a boy stop at once from the commencement of measles, but reappear after convalescence of the disease.

The epileptic aura starts rarely from the tongue. The example of this kind exhibited by a female at the Hospital is on this account interesting. General spasms, accompanied with hemiplegia and contraction of the right limbs, constantly set in, preceded by a sensation of pain exclusively confined to the tongue. The patient, aged 25, presented symptoms of phthisis, and another extraordinary feature of her disease was its supervention upon excessive punishment at the age

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* Lancet, January, 1866.

of 12! A similar though still rarer instance of aura originating from the tongue has been observed by Dr. Radcliffe, in a youth 16 or 17 years old. "The fits were always preceded by a painful sensation, referred to the base of the tongue, and exactly localized in the *foramen cœcum* of Morgagni, at the apex of the V formed by the papillæ vallatæ." *

I will not review the other less unusual forms of epileptic aura, but will close this important subject with the histories of two interesting instances of motor aura. Let me, however, refer to Case VI.—Syphilitic Epilepsy—as evidence of an aura preceding for some time the attacks, and acknowledging a well-authenticated central source.

CASE XX. *Epilepsy upon typhoid fever, with motor aura starting from the right leg.*

W. B——, clerk, aged 28, single, of temperate habits, came under my care in June, 1862. Had typhoid fever in the autumn of 1860, and throughout convalescence, pain and cramps of the right leg, which was inclined to remain most of the time flexed on the thigh. This condition lasted over three months, and it had mostly subsided, when on the morning of March 29, 1861, as he turned over in bed, his right leg was drawn up with cramps; it became rigid, along with the back and neck, and he experienced besides a terrible feeling, which spread from the foot upwards, until he lost consciousness and lay in stupor for about half an hour. Two hours after the first fit a second fit succeeded, initiated by the same cramps and rigidity of the right leg, and he came out of it recognizing every one around him; there was, however, a numbness of the right leg. Otherwise he remained apparently so well that he returned to business the next morning; but seven or eight days after an-

* Clinical Medicine, by Trousseau. Trans. by P. Victor Bazire, M.D. Vol. I. London, 1867, p. 64.—Editor's note.

other fit occurred, ushered in by the above cramp and feeling, and followed by partial lameness in the right leg. Nevertheless he could use the limb and became conscious, and could talk immediately after the fit. From this time the attacks kept on repeating every three or four weeks; he would have them, and would be lame for a day. The cramp would come on suddenly along with a creeping sensation, as if some insects were under the skin, and a peculiar feeling as if the limb was becoming dead from the toes and foot upwards into the leg and body, until it reached the head. He frothed at the mouth, but did not bite his tongue during the spasms. No evidence of impairment of sensibility nor muscular atrophy discoverable in the leg; the bodily condition looked reduced, pale face, pupils contracted, retina normal, anæmic murmurs in the heart, pulse 70 and feeble, respiration 14. Tongue clean, appetite poor, habitual constipation. Restless nights, troubled with insomnia. Urine acid, abounding in phosphates and uric acid. Specific gravity 1029.

It is unimportant to allude to the remedies tried by the patient, unless to say that tightened ligatures below the knee did not succeed in preventing the recurrence of fits, and that large doses of bromide of potassium equally proved powerless against it.

I placed this patient on liquor potassæ arsenitis ♏ v. in infusion of quassia, three times a day before meals, gradually increasing the quantity of the arseniate to eight minims every time. He was further directed to use cod-liver oil, to take good, generous food, with coffee and a moderate allowance of wine or beer at meal-times, and to be packed in a wet sheet every morning and evening. In addition I painted a band three inches wide, below the right knee, with blistering collodion, renewing its application occasionally to maintain a constant counter-irritation to this part. To solace the pain caused by the blister, as well as to avoid peripheral irritation, hypodermic injections of from $\frac{1}{10}$ to $\frac{1}{6}$ gr. of morphine, with $\frac{1}{50}$ gr. of atropine, were made in the leg every night, with the immediate effect of inducing refreshing sleep. The attacks recurred every ten or fifteen days when this treatment was instituted; general health improved, and no fits appeared in three months. But at this time the patient, considering himself cured, discontinued blistering the leg, and the shower-baths, which had been substituted for the packing in the wet sheet. Thereupon a relapse took place, and he had a less severe though identical fit to the former ones. He consequently had to resort to blistering the leg, and to a strict observance of previous directions. Counter-irritation to the leg was then persevered in for a

period of nine months, when he would allow the part to heal for a few days before painting it anew with blistering collodion, and finally with tincture of iodine in its stead. Throughout this second course of treatment he had recourse to the hypodermic injections of morphine and atropine only to allay pain of the blisters. On no occasion did I order bromide of potassium, and the amount of arsenic never surpassed eight minims of the liquor potassæ arsenitis, as beyond this dose it would induce gastric disturbance. No further epileptic attack in 1863 nor in 1864.

Friedberg* describes, under the name of *myopathia dyscrasica*, a lesion consisting of sanguineous extravasation and minute abscesses, with a granular fatty degeneration of the muscular fibres, following typhoid fever. Perhaps such trouble presented itself in the foregoing case during the convalescence of the fever, when the depressed state of circulation was equally propitious to the development of epilepsy, readily provoked by irritation spreading from the local lesion in the muscles of the leg. Be this as it may, it is manifest that the local perturbation of the blisters by changing nutrition at the peripheral origin of the aura, and the tonic treatment by regenerating the blood, sufficed to remove the source of the epileptic attacks.

The following interesting case has been previously brought to notice in a cursory manner, and I now add its detailed account, as contained in a note read by invitation before the Connecticut Medical Convention at Hartford, May 27, 1869, and accompanied with presentation of the excised portion of the ulnar nerve.

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* Pathologie und Therapie der Muskellähmung. Leipzig, 1862.

Case XXI.—*Injury to the right ulnar nerve. Epileptic attacks without unconsciousness, preceded by motor aura starting from the right little and ring fingers. Excision of ulnar nerve above the elbow.*

A girl, fifteen years old, came under my care the 26th of September, 1868. No hereditary taint of nervous disease exists in her family. When an infant had twitchings while teething, and her mother says that she seemed to use the left arm before she did the right one, although she at no occasion presented symptoms of palsy. She menstruated for the first time a year ago last October, and has continued regular ever since. She suffers but rarely during the catamenial period, and the discharge flows freely. In the winter of 1864 she was thrown out of a sleigh, fell on the right arm, bruising it near the elbow, and became instantly seized with cramps of the right limbs. The same but more violent spasms recurred a year after, and have been frequently repeated, extending over the muscles of the right side of the trunk. The paroxysm always burst out with a peculiar tingling sensation and cramp in the right ring and little fingers, creeping up the arm to the spine, thence travelling to the leg, until the whole right side of the body is thrown into a violent tonic convulsion. The right arm and hand are smaller than the left, the difference being hardly manifest between the feet. The right ring and little fingers and the hypothenar muscles were comparatively more atrophied than the rest of the hand, the above-mentioned two fingers remaining in semi-flexion, although they could be extended without effort on the part of the patient. The skin, drier and by three degrees colder in the right than on the left hand, which exhibited a temperature of 93° Fahr., displayed a vesicular eruption and a great roughness in the right fore-arm, with a boil at the posterior and ulnar side of the wrist. The right hand, weaker than the left, felt sometimes uncomfortable to the girl. It was manifest that the muscles in connection with the ulnar nerve were deficient in firmness and in electro-muscular contractility. Tactile sensibility was slightly increased in the right hand, without pain or burning sensation, or change of appearance in the skin of the most affected fingers. Neither was the right hand, usually chilly, in any way more impressed by heat or cold than the left. A difference existed in the tension of the pulse in either wrist, rendered manifest by the tracings of the sphygmograph, and the rhythm of the right radial artery was 89, whereas that of the left 92. Circulation was also completely arrested in the right radial artery during the spasmodic paroxysms.

There was no dissimilarity in the condition of the lower limbs, excepting an occasional feebleness of the right leg upon the occurrence of repeated attacks. No signs of facial paralysis; hearing and sight unimpaired; pupils equally dilated; appearance and blood-vessels of the retina natural; no headache, no loss of smell or taste; tongue slightly coated; appetite poor; habitual constipation; nothing unnatural with the heart and lungs; respiration 18; spine tender between the shoulders; urine acid; specific gravity 1030, charged with lithates and without albumen or sugar. Now, in regard to the attacks, they have increased in frequency and severity since 1865, happening during the day or night, occasionally as many as fifteen or twenty times in succession, and being attended with unconsciousness but once, and that lately. Every paroxysm, whether seizing her awake or asleep, is invariably preceded by the peculiar feeling and cramps of the right ring and little fingers; stretching them out, or running fast when so warned by the aura, checks considerably or prevents the fits. No sooner is the aura announced than the girl turns very pale, her pupils largely dilated, and she utters a cry; at the same time the back and limbs are firmly contracted in tonic spasms, causing trachelismus and great congestion of the face. She does not bite her tongue during the attacks, nor has she ever become unconscious excepting once, as previously stated—last July. Neither has she remained comatose after the fits, which, however, leave her languid and depressed. She has not passed any menstrual period without spasms on or about its end. The application of a moderate induced current to the right ulnar nerve, or to the lower cervical region, did on two occasions respectively determine the convulsive paroxysm with the foregoing symptoms.

Such were the main features of this case when I first saw the girl; and let me briefly notice, before entering into any details as to the course of treatment pursued, that the patient had had an issue put to the lumbar region of the spine, and that she had used bromide of potassium and of ammonium, in addition to several other remedies (cotyledon umbilicus, sulphate of zinc, belladonna, etc.), without any improvement in her condition. I may add that I looked upon the case as one of neuritis, the medulla oblongata being the principal region of the nervous centres consecutively involved upon prolonged incidental irritation throughout the ulnar nerve.

My first step was to fix a strap, like a tourniquet, around the wrist, so that the girl could tightly apply a ligature over the ulnar nerve when-

ever she felt in the fingers the initial cramp of the attacks, and to cauterize with a red-hot iron the lower part of the cervical region. The burning determined an immediate change in the temperature, with more comfortable feeling of the right hand, and the operation was renewed three times within a month. The girl further practised gymnastics, and used hydrotherapics and other proper means to improve the nutrition of the atrophied limb, together with a generous diet. Arseniate of potash and large doses of ext. conii (gr. viii) with ergotine, were the only internal remedies employed in addition. The attacks decreased in frequency and severity: but the ligature, at different times applied around the wrist or above the elbow, failed to arrest the fits. The girl opposed herself strenuously to more burning of the neck; blisters were therefore substituted, and finally a silver-wire seton was inserted at the lower part of the cervical region. Greater objection still was met with on the part of the girl to caustic or blistering applications around the arm. The above-described plan was persisted in for nearly six months without any evident progress towards cure, and to the great discouragement of the girl. There remained yet to be tried section of the nerve, although I did not feel positive as to the efficiency of the operation, lest neuritis should be propagated upwards much beyond that portion of the ulnar nerve close to the elbow. The parents of the girl were distinctly informed of my apprehension, and the patient decided to submit herself to the trial rather than continue the victim of such distressing fits.

Nerve excision was, therefore, carried out on the 22d of April, with the kind assistance of my friend Dr. John H. Douglas. At the request of the parents chloroform was used instead of ether to produce anæsthesia—the girl of course being duly prepared for the operation. An incision, about three inches long, from the middle of the interval between the inner condyle of the humerus and the olecranon, and obliquely running across to the middle of the inner side of the arm, exposed the ulnar nerve. On dissecting the portion concealed under the heads of the flexor carpi ulnaris, the girl had three very noticeable spasmodic seizures. At this site the nerve displayed a deep brick color; once isolated from the elbow upwards, I divided it above, where it appeared normal, and removed nearly two inches of the nervous trunk down to its passage under the flexor carpi ulnaris. There was a little hemorrhage, easily arrested: the edges of the wound were then united by three silver sutures, and an ice-bag was applied to the wound. The effects of chloroform passed away without unpleasant symptoms; the girl rapidly recovered

consciousness, crying and complaining of pain and numbness in the two smaller fingers and ulnar side of the hand. After the operation the temperature of the right hand was 98°, that of the left not reaching 93°. The pulse in the right wrist became also fuller. The operation was performed at noon, and in the evening the pain in the hand subsided considerably. Sensibility had completely disappeared from the inner side of the ring finger, from the little one, and the inner dorsal and palmar regions of the hand. Curious enough, the tip of the little finger, very red, retained tactile sensibility, and has to this day preserved it. Motion of the two fingers did not seem much more impeded after than before the operation. The incision healed without discharge; the silver sutures were removed on the 25th, three days after the operation, the wound being then painless, and the heat of the right hand 94°—hardly one degree higher than that of the left. The ice became disagreeable to the girl, and was in consequence discontinued the fifth day after the operation; thereupon the superficial parts of the cut separated from each other, and for two days discharged clear serum. No change occurred during this time in the hand, but the wrist-joint felt sore. Pressing on the wound did not awake any painful feeling; however, if the pressure were gently exerted over the end of the centripetal portion of the ulnar nerve, the hand felt as if numb. This effect could not be produced by trying compression over the extremity of the distal end of the ulnar nerve. The girl continued taking hemlock and ergotine as before, and with the seton still inserted in the lower part of the back of the neck.

Little less than two inches, by measurement, were removed from the ulnar nerve-trunk, the thickness of the nerve being two lines at the lower point of section, where the nerve sinks beneath the flexor carpi ulnaris, while the upper section did not measure one line in diameter, and seemed normal. The nerve was semi-transparent, as remarked before, and had a brick-red color at the elbow, the discoloration gradually fading upwards to about one inch below the point of the upper division. The neurilemma was thickened by hyperplasia of connective elements and nuclei, giving the red, pellucid aspect to the nerve. A few fatty globules appeared intermingled with these elements. The capillaries of the cellular sheath were enlarged, with granular walls. The nerve-fibres, in many places reduced to their external sheath, displaying small oval nuclei, were in several others distended by a fine fatty granular mass of altered myeline. This latter degeneration did not exhibit itself on specimens prepared from the apparently normal portion of the

nervous element, but in them the nerve-fibres were pressed by the already described multiplication of connective elements.

I would not like to venture any prognosis as to the final result of this case; but I dread, upon microscopical examination of the removed portion of the nerve, that neuritis, not stopping its untoward propagation along the inner cord of the brachial plexus, might induce the recurrence of the attacks and extensive paralysis upon the consecutive degeneration of the spinal cord. To this date (May 25, 1869) no trouble has interfered with the progress of the case. The question prompts itself, however, why could I expect any benefit from the operation, suspecting the existence of neuritis propagata? To this I answer, for more than one important reason. There was no permanent loss of power nor atrophy in the right lower limb, its transient weakness after the attacks being naturally accounted for by exhaustion, and not degeneration of the spinal cord. The painless state and limited atrophy of the hand predicted perfect success from nerve-excision, which could achieve a twofold effect; first, to arrest the attacks by cutting off the passage of peripheral irritations through the ulnar nerve to the nervous centres; and second, but not less important, by equally preventing threatening impairment of structure in the very nervous centres in consequence of persistent morbid reflex action through nutritive nerve-fibres. Experience shows that neuralgia, or algesia, supervening upon injury to a nerve, is seldom if ever permanently cured by division of the affected

nerve. Cases reported by Azam, Richet, Mursick, Gherini, Carnochan, and the authors of the interesting work "On Gunshot Wounds and Injuries of the Nerves," bear evidence to this assertion. Had pain, or burning sensation in the hand, or neuralgia, existed along with the epileptic fits, then I should not have undertaken the operation, because these symptoms would have, as usual, recurred upon excision of the nerve. I am further aware that in a case similar to the one now related, and described by Sir E. Home,* section of the nerve proved unsuccessful in removing the spasmodic fits: "A gentleman received a violent sprain of his thumb by the weight of his body being thrown upon it in saving himself, when nearly thrown off by a sudden motion of his horse. He was afterwards liable to paroxysms, in which his thumb was first bent in towards the palm of his hand; a spasm then took place in the muscles of the arm, after which he became insensible, and continued so for about a quarter of an hour. The attacks returned frequently in the arm; but it was found that the pressure of a tourniquet prevented the insensibility. A nerve in this case was divided without success. The tourniquet lost its effects in arresting the spasms, and he died suddenly after three months, but there was no examination of the body." In this interesting instance the train of symptoms were not entirely like those of the girl I operated upon, for she never but once re-

* London Phil. Trans., 1801, and Mayo's Outlines of Human Pathology. London, 1836, p. 143.

mained insensible after the spasms; moreover, reunion might have rapidly taken place upon simple section of the nerve, or, what is a great deal more probable, the nerve divided by Home was not the affected one; for, besides the external branch of the median nerve which supplies the abductor, opponens, and outer head of the flexor brevis pollicis, there is another equally important deep palmar branch of the ulnar nerve distributed to the adductor and inner head of the flexor brevis pollicis, and on this account the case of Sir Everard is open to objection concerning the merits of the operation.

I cannot find a satisfactory explanation to the persistence of tactile sensibility in the tip of the little finger, after such a complete section of the ulnar nerve, unless we admit a digital branch of the median nerve supplying this region. I know that division, or even excision of an extensive portion of a nerve is not necessarily attended with irreparable loss of sensibility or motion. It would not matter how the nervous influence acts in such circumstances; but if it had reached in this case the lower portion of the ulnar nerve across the extensive gap at the elbow, tactile sensibility could not have been more manifest at the tip of the little finger than throughout the other cutaneous regions supplied by the nerve, and yet remaining insensible.

Has epilepsy any immediate influence on coincident paralysis, or is this a symptom of accidental origin? Reynolds holds that the character of the association between epilepsy and paralysis is accidental and not

consecutive; but the inferences from the cases under consideration—in which paralytic symptoms occurred subsequently to, and in direct relation with the epileptic paroxysms—lead me to a quite contrary opinion to that of the eminent English physician. Thus, paralytic symptoms are distinctly recorded in the following of the 306 patients:—

	Males.	Females.
Right hemiplegia, permanent	3	6
Right hemiplegia, temporary		3
Right hemiplegia and cardiac disease		1
Right hemiplegia and aphasia		1
Left hemiplegia, permanent	7	10
Left hemiplegia, temporary	1	1
Left hemiplegia and cardiac disease		1
Left hemiplegia and loss of speech		1
Left hemiplegia and right facial paralysis		1
Paraplegia, permanent	2	3
Paraplegia, temporary	1	
General paralysis and cardiac disease		1
Tremor and palsy		1
Paralysis of the tongue		1
Facial paralysis and convergent strabismus		1
Strabismus	1	
Paralysis of the face (right side) and of left arm		1
Ptosis	2	
Blindness and loss of smell	1	
Deafness	1	1
Loss of smell		1
Aphasia	1	
Speechlessness	1	2
Paralysis of right arm		2
Paralysis of left arm		1
Paralysis of right arm and eye-lid	1	
Anæsthesia of right limbs		1
Atrophy of right arm		1
Contraction of left limbs	1	

	Males.	Females.
Contraction and atrophy of left arm	1	
Contraction of left arm and right facial palsy	1	
Contraction in right limbs		1

One single case of petit mal in the male sex was attended with hemiplegia on left side; whereas the two forms of attacks combined induced more or less extensive paralysis thrice among males and seven times among females, of whom one, however, exhibited anæsthesia of right limbs with diminution of sight and hearing on the same side, without any loss whatever of motor power. The preponderance of left hemiplegia is quite conspicuous, it being interesting to find that positive signs of cardiac disease existed only in one case of right hemiplegia and in another of left, both exclusively in females. These cases do not uphold the statement of J. Rengade and L. Reynaud, that hemiplegia is more frequently observed in the right than in the left side, as a consecutive accident of epilepsy.* Nor do they bring much light on the important question as to what chain of causation might associate the primary lesions of the left cerebral hemisphere originating right hemiplegia, with the obscure condition of aphasia. I would not venture, therefore, to throw out any consideration over a subject which has to be elucidated not with clinical but with anatomo-pathological facts. Whatever may be the mode of connection, it is evident that a coincidence has been found between right hemiplegia and aphasia

* Recherches Statistiques sur les Accidents Produits par l'Accès Epileptique. Extrait de la Gazette Hebdomadaire, Paris, 1865.

in those instances where the left third frontal convolution appeared damaged; but to assert from this that such limited cerebral convolution ministers to the faculty of speech is too absolute a conclusion. We have as yet forgotten, in our eagerness to establish a theory, to consider the no less important value of negative examples, which begin already to outweigh the arguments on behalf of the exclusive function assigned to the third left frontal convolution. Consequently, for my own part, I am still unprepared to accept a necessary connection between lesion of the just named convolution and aphasia, which may be an attendant of trouble in other regions of the brain and Pons Varolii, and which has furthermore failed to be constantly present in instances of alteration of the third left frontal convolution, as I have tried to show when treating of the Pathological Anatomy of Epilepsy.

The ophthalmic arteries are the main channel of communication between the cerebral arteries and those of the outside of the head. The derivative circulation of the brain takes place, as shown by J. P. Sucquet,* in that portion of the facial integuments, nose, forehead, cheeks, and conjunctiva, supplied by the orbital group of the ophthalmic artery. Hence the paleness of those parts at the inception of the epileptic fit, and their lividness from capillary paralysis after the paroxysm is over; hence, again, the flushed face of maniacs, or of those with cerebral softening, whose

* D'une Circulation Dérivative dans les Membres et dans la Tête chez L'homme. Paris, 1862.

conjunctivæ are usually hyperæmic, and the eye-lids the seat of a peculiar muco-purulent discharge. The state of the intra-cranial circulation is, undoubtedly, reflected on the retina, but the vascular condition of this latter may become frequently modified, the changes arising therefrom being referred to absolutely local mischief; whereas it is easy to understand also, that the subtle and shifting functional impairment of the capillary circulation may and does occur in epilepsy, without leaving any ulterior trace in the retina or brain. Furthermore, in a case of wound of the spinal cord, in which there were, on the left side, symptoms like those following section of the cervical sympathetic,—*i. e.*: ptosis, contraction of the pupil, neuralgia in most of the branches of the fifth nerve, hyperæsthesia to touch, and perspiration upon the least exertion made by the patient,—Dr. Hughlins Jackson could not find the least difference in the size of the vessels or in the color of the optic disc; besides there was not, nor had there ever been, any defect of sight whatever.* Moreover, I have often seen an induced current applied to the cervical sympathetic, as well as the galvanic current—capable of originating epileptiform convulsions, as discovered by Callenfels —to cause retinal anæmia at first, and soon after hyperæmia, which ceases upon discontinuance of the current. This, and the foregoing facts, account for why departure from a healthy vascular condition of the retina is not detected in every case of epilepsy.

* Medical Times and Gazette. Oct. 3, 1863, p. 359.

Bouchut, in his valuable work on the Diagnosis of Nervous Diseases by the Ophthalmoscope, states: that with some epileptics there is either vascular anomaly in the retina—*angioplania*, or increased number and size of the retinal veins—*retinal hyperangia*. This statement is advanced with reserve, and, avoiding to pronounce himself on the subject, Bouchut besides says: "Be this as it may, excepting those cases of epilepsy consequent on cerebral lesion, powerful enough to congest the encephalon or to impede the circulation of the cerebral sinuses, thereupon inducing complete or partial serous infiltration of the optic disk—epilepsy does not bring about beyond a considerable hyperæmia, or hyperangia (multiplication of blood-vessels), and in some instances what I have called angioplania, that is, an anomalous distribution of blood-vessels in the fundus of the eye. This is, undoubtedly, insufficient to diagnosticate epilepsy; yet with some epileptics the hyperæmia of the retina may be sufficient to make us foretell a threatening fit, or to detect the traces of one which has already passed."* Bouchut adds, that Dr. Duguet made in 1861 numerous ophthalmoscopic examinations of the epileptics at the Salpêtrière, in the division of Dr. Moreau. In a series of thirty-one cases, nine exhibited no unnatural appearance of the retina, though in some of them epilepsy was associated to unilateral atrophy of the brain, idiocy, imbecility, &c. Of the

* Du Diagnostic des Maladies du Système Nerveux. Paris, 1866. p. 377.

other twenty-two,' a few had congenital cataract, or iritis, or atrophy of the eye-ball, whereas the retina in the remaining displayed signs of atrophy of the optic disc, or congestion with patches of exudation, and other evidences of choroiditis, apparently connected with the cerebral trouble. "A great number of other epileptics—at least 180—were also examined several times with the ophthalmoscope by Dr. Duguet, and in none was any lesion of the optic nerve or of the retina detected." T. Clifford Allbutt* examined the optic nerve and retina in forty-three cases of insanity dependent on epilepsy, and found—in fifteen disease of the optic nerve or retina; nine were doubtful, and nineteen showed no change. Simple epilepsy is not commonly followed by disease of the optic nerve. In most of the cases presenting optic changes, organic disease was known to exist from other symptoms.

Dr. Hughlins Jackson examined the retina during a fit of epilepsy. He found the optic disc whiter than normal, and the veins were large and dark.† In two cases in which the patients complained of headache with a little dimness of sight after a paroxysm of epilepsy, Dr. Jackson observed in one the veins remarkably large and dark, and the arteries also seemed darker than normal. The disc was hyperæmic. The examination took place two and a half hours after the fit. In the second instance, after the fit, the veins appeared very large and the optic disc was

* British and Medical Journal. March 14, 1868.

† Medical Times and Gazette. Oct. 3, 1863, p. 359.

reddened as if flecked with red. It was not uniformly colored, but somewhat like white bibulous paper slightly smeared by red ink.

As to myself, I have examined with the ophthalmoscope most every epileptic under my care for the past five years. I have generally used for the purpose the ophthalmoscope of Galezowski, or that of Follin and Nachet, which renders the examination of the eye very easy. I have never succeeded in examining satisfactorily the retina during a fit. I have often observed it eight or ten minutes after, and even while the patient was partially comatose. I have likewise examined the eye as the patient experienced all the precursory symptoms of an attack; under these latter circumstances I have often, but not always, noticed the optic disk paler than normal, and the arteries indistinct, while the veins remained distended and darker. After the fit, the fundus and the optic disk have appeared hyperæmic, but not constantly so; in not very rare instances the retinal hyperæmia has been, indeed, insignificant. I have not been able to make out frequently the multiplication of blood-vessels pointed out by Bouchut, but I have often remarked a tortuous and irregular disposition of the blood-vessels and their monoliform distention. Rarely have I found any extravasation of blood as consequence of the fits. Changes in the vascular elements have been constant where sight has remained impaired after the fits, or where these were associated to strabismus, nictalopia, hallucinations,

or any other functional derangement of the eye. In some instances where, without any paralytic trouble, the fits have been followed by dilatation of one of the pupils, with hyperæmia of the conjunctiva, I have not discovered any unhealthy condition of the optic disc or of the retina, nor any difference with that of the opposite side.

Judging from my own observation, the hyperæmic condition of the retina in uncomplicated cases of epilepsy does not last longer than twenty-four hours, and it generally disappears as soon as the patient has completely recovered from the after-effects of the fit. I, of course, allude to any hyperæmia sufficient to indicate the previous occurrence of a fit, as I deem it almost impossible to distinguish the slight degrees of hyperæmia from the normal circulatory condition of the retina. Let me further state that, it is by no means uncommon to meet with epileptics laboring under epileptic insanity, the immediate result of a fit, and whose eyes would not exhibit any appreciable evidence of their mental derangement. I need not state that most of the results here exposed have been controlled by the different Assistant Physicians to the Hospital. I may also add that, in 1866 Dr. Henry D. Noyes examined with the ophthalmoscope, on three different occasions, the epileptics then at the Hospital. The notes of such competent examinations taken respectively by Drs. Castle, McClung, and Edwards agree with those given by Duguet and Bouchut, with my several observations, and plainly evince—that the re-

tina does not undergo an invariable constant change in epilepsy; that this latter may exist in its worst forms without bringing about any apparent modification of the former. Consequently, the alterations of the retina that might accompany some cases of epilepsy are accessory to the disease, and have no specific diagnostic value.

I lack a sufficient number of facts to establish any causal relation between cardiac disease and epilepsy, and need not repeat what I before remarked on the subject. The signs of cardiac lesion are noted in three out of 130 males: one of them presented hereditary taint of epilepsy, and the two others impediment to articulation. In one of them, with mitral regurgitant murmur, thick speech, left pupil larger than the right, there was ground to suspect cerebral embolism, favored in addition by the patient's inveterate habit of intemperance, it being a well-known fact that the generation of clots in the circulatory system is frequently induced by intemperance. The complication with disease of the heart reaches a higher degree among females, of whom fourteen out of 176 exhibit unequivocal signs of valvular lesion, and three of them irregular action of the heart. In five of the first group and in one of the second hereditary predisposition to epilepsy showed itself; the first group including still another patient, the offspring of consanguineous marriage between first cousins. Paralytic and cardiac symptoms coincided in five of the above fourteen cases—namely, one with general pa-

ralysis, one with right hemiplegia, and three with left hemiplegia. I shall not speculate on the agency that disease of the heart might have exerted on the development of paralysis in each of these instances, because on no occasion have the facts manifested, directly or indirectly, any close association.

A glance at the Synoptic Tables in Chapter III. will convince us of the depressed state of circulation and feeble condition of the pulse, which, taking the average of epileptic cases under my care, has ranged above the normal frequency. Different tracings obtained with the sphygmograph lead me to assert—that there is an increased development with acceleration and marked dicrotism of the pulse prior to and for some time after the epileptic attacks, whereas the

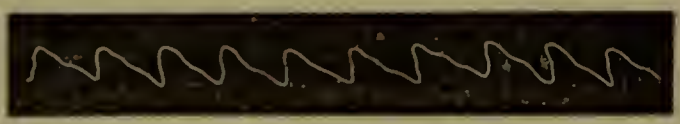


Fig. 1.

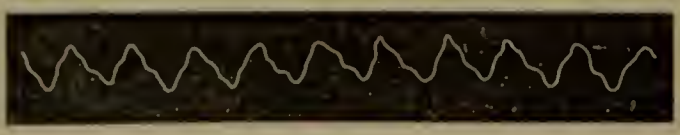


Fig. 2.



Figs. 3 and 4.



Figs. 5 and 6.

arterial tension and frequency of the beatings, which also become irregular, decrease during and immediately after the paroxysm. The first of these conditions may be discovered from two to six minutes before the outbreak of the fit, while it may persist for two or three hours after cessation of the spasms, as indicated by the repeated observations I have made on the subject. Fig. 1 shows the tracing of the normal pulse in a male epileptic aged 32. Fig. 2, the tracings about two minutes before a fit. Figs. 3 and 4, the pulse during the stage of coma, and Figs. 5 and 6, the pulse when the patient had partially recovered consciousness, about ten or fifteen minutes subsequent to the fit. The sphygmograph I have employed is that of E. Groux, with a very ingenious clock-movement to obtain the tracings.

An important phenomenon in regard to respiration in epilepsy, which, so far as I know, has not been hitherto remarked, is the loss of its normal relation to the circulatory activity. It is a physiological fact that the pulsations and the respiratory movements increase simultaneously, acceleration of the pulse being consequently attended with that of respiration. L. V. Marcé noticed that in melancholy, and especially in melancholy with stupor, the respiration and circulation become deranged as to their rhythm and relative frequency. The same derangement I have observed with epilepsy, and in such a constant manner as to convince me of its being an important peculiar symptom of the disease.

The average normal rate between the number of pulsations and respirations has been determined, after extensive researches by Marcé,* to be as follows:—

To	43	pulsations correspond	16	respirations.
"	53	" "	19	"
"	70	" "	24	"
"	82	" "	24	"
"	104	" "	35	"
"	142	" "	42	"
"	172	" "	50	"

I have repeated the investigations made by Marcé, and recorded at different times the number of respirations and the pulse in 108 individuals not troubled in any way with the respiratory organs, and enjoying good health. The results are as follows, giving the maximum and minimum of the number of respirations:—

To	65	pulsations	16—21	respirations.	Adults.
"	68	"	19—24	"	
"	70	"	17—20	"	
"	73	"	19—23	"	
"	75	"	18—22	"	
"	80	"	20—23	"	
"	85	"	21—24	"	
"	90	"	26—28	"	Infants.
"	98	"	30—	"	
"	104	"	32—	"	

* Archives Générales de Médecine, 1855, and Traité Pratique des Maladies Mentales. Paris, 1862, p. 321.

I must remark that in the majority of instances the higher proportion of the respiratory movements was met with in females. Considering either of the foregoing tables as indicating the ordinary relation of the pulse and respiration, we may be at once struck by the disproportion in the following statement, the second part of which has been recently drawn by Dr. J. H. Morgan, Assistant Physician to the Hospital for Epileptics and Paralytics.

Patients under no treatment:—

Age.			
Males.	Females.	Pulsations.	Respiration.
18		92	13
	22	102	20
26		84	16
32		80	20
15		94	21
	16	98	18
	10	104	22
	5	108	19
40		86	14
22		95	19
	18	88	16
26		96	24
	15	98	18
38		84	12

Patients who have been little or not at all benefited by treatment at the Hospital:—

Age.			
Males.	Females.	Pulsations.	Respiration.
	21	92	21
	17	95	21
	32	100	15
	21	80	24
	31	84	16
	20	80	14
	20	92	16
	29	100	18
	23	90	14
	23	90	26
	25	88	20
	?	94	21
	28	75	21
	25	96	20
	35	69	16
	15	80	21
	20	80	19
	20	94	22
	18	100	24
	28	105	28
	32	64	21
?		84	24
39		82	18
14		90	20
19		90	15
24		94	22

It will be easy to account for the coldness and

lividness of the extremities—so striking with epileptics—by the above manifest slowness of the respiratory activity, which is certainly due to the circulation of blood imperfectly decarbonized upon the insufficient respiration here noticed.

Unnatural perspiration has been noted on three occasions among the males, and on two among the females, directly connected with the fits. In one of the males with petit mal, perspiration in the head and arms preceded the attacks. Another, also with petit mal, perspired profusely until becoming exhausted after the fits. A third, epileptic after fracture of the skull, causing blindness and loss of smell, perspired freely and very offensively after the paroxysms. Offensive perspiration could be noticed equally in a female, epileptic from uterine trouble and dysmenorrhœa. In all these instances a variable degree of cutaneous hyperæmia was present along with perspiration. Not uncommonly a peculiar odor is perceived about epileptics, even when perspiration does not increase before or after the fits. This phenomenon is not so rare that it could not be observed among hospital patients, and bears great analogy to the peculiar odor exhaled by patients in the last stage of some cerebral diseases, although it is by no means of such unfavorable character. Tissot* observed in two of his patients an unbearable cadaveric odor ; and McDonnell†

* Traité de l'Epilepsie, p. 5.

† The Dublin Quarterly Journal of Medical Science, No. LXIII., February, 1864.

mentions the case of a lad 18 years of age, epileptic, dull, stupid, and slow of speech; who "had a peculiar odor exhaled from the skin, like a mixture of garlic and brass." With the exception of these I have found no other references to the above-described symptoms.

A general petechial eruption in the face, neck, and limbs supervened upon the epileptic paroxysms in the following case: A female, 30 years old, had petit mal in daytime and spasms at night, the former constantly succeeded by fits of laughter and mania. Every nocturnal paroxysm, repeating once or twice a week, left a minute confluent eruption of the neck and upper limbs, that passed off within two or three days, and which was more pronounced if the fits of petit mal recurred immediately upon the nocturnal attacks. A similar to this instance is reported by Chatelet. A man with frequent fits of epilepsy had a petechial eruption with paroxysms of furious mania that yielded to treatment, the epilepsy, however, still continuing. The eruption displayed itself in the face, neck, and shoulders previous to the maniacal excitement, and upon repeated spasmodic attacks, but never upon isolated ones, nor after a series of spasmodic fits several days apart from the preceding.* Parrot† reports the case of a hystero-epileptic female for many years subject to bloody sweating of the knees, thighs,

* Mémoires et Comptes Rendus de la Société Médicale de Lyon. Tome II. 1863, p. 199.

† Etudes sur la Sueur de Sang. Gaz. Hebd. de Paris, 1859, p. 40.

chest, lower eyelids, hands and face, without reddening of the skin on these parts. The tears became once discolored with blood, and on another occasion the skin of the hypogastric region perspired blood during one of the frequent attacks of hematemesis attended with violent epigastric pain. Such harmless hemorrhages never occurred unless after mental emotion and along with complete motor and sensory paralysis; at the time of menstruation—which kept on undisturbed—these neuropathic cutaneous hemorrhages ceased. Parrot further states that their production in this and other similar instances is connected with localized phenomena of pain and spasms.

The skin of my patients, in addition to the peculiar sallowness of the face, has sometimes displayed herpetic or vesicular eruptions, to which reference has been already made, as evincing the peripheral derangement of the vaso-motor nerves. These eruptions usually appear in the trunk and limbs, exhibiting a sort of symmetrical location not rarely corresponding to the course of some of the nerves. On less frequent occasions, the eruption has been seated around some of the external apertures, mouth, eye-lids, prepuce, etc. The temperature of the hands and feet in several of my patients—measured by the thermometer—has been one or two degrees below that of the rest of the body. I have not recorded any variation from the usual temperature in many patients I have examined during the fits, but the temperature in all the cases has been manifestly below the normal standard.

The urinary secretion presents important changes in connection with the epileptic attacks; in a male, whose history will be hereafter related, poliuria supervened on traumatic injury to the head. A girl, aged 23, with nocturnal paroxysms of unknown origin since the age of 19, was also diabetic. In another whose case is detailed page 217, I found sugar in the urine after the fits. This is the only example of the kind I have met with, contrary to the statement of Reynoso,* who regards the phenomenon as an ordinary sequel of epilepsy—a view further disproved by researches of Michea and other authors. I never discovered albumen in any of my cases.

In order to ascertain the modifications produced on the quantity and quality of the urinary excretion by the epileptic attacks, the following investigations were instituted. Four male adults, three of them with diurnal and the fourth with nocturnal spasms, regularly occurring every day, were selected and kept without treatment for two weeks—their diet consisting of bread, beef, potatoes, rice, milk, and about one and a half pints of coffee, without further allowance of stimulants, nor any food taken between the meals. The experiment was made in April—the average temperature of the weather being 68.34. The change in the quantity and constituents of the urine irregularly and slightly varied from one to another day, and the average quantity and quality of the urine daily passed during the fourteen days by each of the patients was :—

* Annales Médico-Psycologiques, 1852.

QUANTITY OF WATER.	SP. GRAVITY.	UREA.	CHLORIDES.	PHOSPHATES.	REACTION.
C.c.		Grammes.	Grammes.	Grammes.	
940	1020	35.080	18.73	1.062	Acid.
870	1011	31.720	14.38	0.930	Acid.
1230	1016	37.370	20.64	0.984	Acid.
1120	1030	30.150	17.85	1.122	Acid.

No albumen or sugar was detected in any of the cases. The patients were then ordered twenty grains of bromide of potassium in half an ounce of infusion of calumbo, and took a shower-bath in the morning and evening. The fits during another period of fourteen days occurred, namely: twice in the first, once in the second, none in the third, and once in the fourth patient, with nocturnal attacks. The results were:—

	QUANTITY OF WATER.	SP. GRAVITY.	UREA.	CHLORIDES.	PHOSPHATES.	REACTION.
	C.c.		Grms.	Grms.	Grms.	
FIRST PATIENT.						
3 days before 1st fit..	1100	1018	29.130	21.40	0.964	Acid.
24 hours after 1st fit..	1420	1018	27.84	29.15	0.170	Acid.
5 days between 1st and 2d fit.....	1510	1014	27.375	18.75	0.152	Acid.
24 hours after 2d fit..	1830	1016	34.65	23.03	1.098	Acid.
8 days after 2d fit...	1470	1014	28.15	17.84	0.981	Acid.
SECOND PATIENT.						
9 days before 1st fit..	1875	1017	28.75	12.42	1.475	Acid.
24 hours after fit.....	lost.					
5 days after fit.....	1950	1012	18.56	12.63	1.262	Acid.
THIRD PATIENT.	1290	1018	19.66	14.32	0.926	Acid.
FOURTH PATIENT.						
5 days before fit.....	1160	1025	37.47	18.35	0.991	Acid.
24 hours after fit.....	2010	1022	34.92	14.38	1.740	Acid.
9 days after fit.....	1185	1022	22.69	20.71	1.034	Acid.

I again examined in October, 1866, the urine of two females, aged respectively 20 and 27, subject one to weekly paroxysms, and the other once every three weeks. Both took three times daily half a drachm of bromide of potassium, with five minims of Fowler's Solution in tincture of sumbul and water, and besides six grains of ext. conii with two grains of ergotine, morning and night. They used a plain diet, with coffee and no alcoholic stimulants, and the results were :—

	QUANTITY OF WATER.	SP. GRAVITY.	UREA.	CHLORIDES.	PHOSPHATES.	REACTION.
	C.c.	Grms.	Grms.	Grms.	Grms.	
FIRST PATIENT.						
15 days before fit....	1579	1018	35.71	13.80	1.052	Acid.
24 hours after fit....	2290	1016	39.26	11.18	1.194	Acid.
3 weeks after fit....	1084	1016	36.47	10.40	1.064	Acid.
SECOND PATIENT.						
8 days before fit....	1790	1021	21.255	13.10	1.028	Acid.
24 hours after fit....	2930	1016	24.800	12.35	1.123	Acid.
4 days after fit.....	1650	1016	20.462	15.90	0.934	Acid.
24 hours after fit....	lost.					
8 days after fit.....	1870	1014	20.528	20.15	0.961	Acid.
24 hours after fit....	2410	1017	22.899	15.60	1.016	Acid.
4 days after fit.....	1800	1010	19.600	11.53	1.012	Acid.
24 hours after fit....	2020	1010	23.325	14.58	1.914	Acid.
6 days after fit.....	1972	1012	18.180	16.37	0.693	Acid.

The foregoing tables show that the urinary secretion is increased after the paroxysms, as also the quantity of urea, which, in every case, appears above the mean of the days without fits. With one single exception, the proportion of phosphates became increased after the attacks; no regular variation, how-

ever, took place with chlorides, which either augmented or diminished after the fits. The results further evince that the quantity of urea decreases very notably upon exhibition of bromide of potassium, a fact agreeing with the observation made by Professor Bartholow of Cincinnati. I need not add that bromine was in every instance detected combined with the alkalies in the very first urine excreted after its exhibition. Uric acid and deposits of the urates were not very frequent in patients taking bromide of potassium, yet uric acid seemed more constant on days after the paroxysms than at any other period. These results are in accordance with statements made by Pritchard* and E. A. Parkes,† and they have received additional confirmation in more recent researches instituted by I. W. Gibson, on the condition of the urine in three cases of epilepsy, respectively aged 21, 31, and 49. From his examination Gibson states that "there is no constant change in the urine, although there appears to be some connection between the occurrence of the fits and increase in the water and urea, and this increase is subsequent to the fits."‡

* On Diseases of the Nervous System. London. 1822 p. 89.

† The Composition of Urine in Health and Disease. London. 1866. p. 313.

‡ Medico-chirurgical Transactions of London. Vol. L. 1867. p. 75.

CHAPTER VI.

TREATMENT—TREPHINING FOR THE RELIEF OF EPILEPSY —EPILEPTIC INSANITY.

Epilepsy, like every malady, runs its untoward course in a more or less regular manner. Peculiar disorders characterize its various stages, calling different parts of the nervous system into concurrent but not simultaneous action. Their concatenation does not either follow an invariable order; hence the absence or transmutation of certain symptoms, occasionally observed with epileptics. Nor does removal of the originating cause suffice to achieve the cure of epilepsy. There exists another essential element of the disease, *i. e.*, the epileptic habit, that must be previously annihilated in its baneful influence. Then again, the prominent share taken by the circulation in the development of epilepsy renders necessary to its treatment to regenerate the blood, for upon its derangement and consequent disturbed nutrition the excitability of the nervous centres is carried to a disordered degree and the epileptic habit effectually induced. Therefore, anæmia, or any other condition attended with an impaired quality of the blood, ranks ahead in the causation of epilepsy. For all these reasons, it would be assuredly too narrow-minded to rely on the efficacy of any of the so-called anti-epileptic remedies rather than on the more

scientific and fruitful knowledge of the etiology of the disease, to establish the rational bases of therapeutics which ought to counteract chiefly the physiological influences operating on every individual instance. If to this we add, that most popular remedies prove worthless, or even noxious, when promiscuously administered, and that in the etiological range circumstances of the most diverse kinds are capable of superinducing epilepsy, it will be obvious how much these circumstances would prevent fruitful pursuit of the same course of treatment in every case of epilepsy. Having premised these remarks, I will now relate some of the most important instances where the epileptic attacks have been cured, with comments on the means of treatment therein adopted.

Case XXII. *Epilepsy from intemperance. — Hypodermic injections of strychnia.—No recurrence of fits in six years.*

G. C——, a mercantile agent, native of California, aged 21, consulted me March 20, 1864. He had freely indulged since the age of 18 in drinking, without ever suffering from delirium tremens, but frequently becoming intoxicated. No hereditary taint of nervous disease in his family. The first fit took him suddenly on the night of November 9, 1861, after a debauch, and he had several other seizures throughout the week following. Was cupped on the neck, and very much purged with calomel upon this attack. The fits stopped for five months; but as he resumed the former intemperate habits, the paroxysms recurred with greater frequency and violence, and continued, notwithstanding the strict temperance subsequently observed by the patient. The intervening period between the fits seldom extends now beyond four days; and they are always preceded by tinnitus aurium, and occur as much during the day as in the night. There is habitual constriction around the head, with pain extending down along the spine; memory is enfeebled; the limbs are weak and cold, with slug-

gish circulation; they do not appear materially affected as to sensation. Urine high-colored, acid, and scanty. No albumen nor sugar. Thick deposits of phosphates and urates. Specific gravity 1035. Pulse 95, very weak, and irregular. Respiration 17. Cardiac action feeble; no unnatural murmur. Appetite poor. Tongue slightly furred. Bowels inclined to costiveness. He is very restless at nights, sleeps very little, constantly awaking in the morning with giddiness and pain at the occiput. The fits, as already stated, are preceded by a peculiar ringing in the ears for two or three hours, or even one day, along with an indescribable strangeness or cloudy condition of the mind, and he goes to sleep heavily after the paroxysms. He has used indigo, sulphate of zinc, nitrate of silver, and other preparations, without improvement. He tried the nitrate of silver until it determined dyspeptic trouble, and slight discoloration of the skin, still persisting when I saw him.

He was ordered an easily digestible and nutritious diet; cod-liver oil; cold sponging, followed by friction of the body every morning and evening; out-door exercise, and one tablespoonful three times daily of the following mixture:—

℞ Potassi bromidi, ʒ iv.
Potassi iodidi, ʒ j.
Tinct. cardam., f. ℥ j.
Aquæ pimentæ, f. ℥ v.
Misce.

April 20. Had one fit yesterday, the previous having occurred the 22d of March, this being the longest period during the last eighteen months that he has remained without any attack. Has slept better, awaking in the morning free from headache or giddiness. The tinnitus aurium preceded for a few moments only this last attack, which occurred early in the morning, after getting up. The circulation of the limbs continues very deficient, and they are uncomfortably chilly. The dose of bromide is increased in the above mixture, so that he takes thirty grains three times a day. One-ninetieth of a grain of strychnia is injected in each leg every day, with immediate relief and pleasant feeling of warmth.

Twenty-two consecutive injections of strychnia were repeated in the same number of days and then stopped, the limbs no longer continuing chilly or livid. The general appearance of the patient and his appetite greatly improved. Pulse 75, of fair strength; respiration 18. No alteration made to the treatment.

In the night of July 28 he had an epileptic fit, without premonition whatever. Three days after, while at breakfast, he became dizzy and lost consciousness, with no other symptoms amounting to a fit. I did not see him until the 4th of August. The bromide was carried to forty grains three times daily, still combined with the same dose of iodide of potassium. He was further directed to have four grains of extract of conium and two of ergotine, in the morning and evening; to substitute a short shower-bath for the sponging, and to go to the country.

From this date no return of fits reported by the patient, who nevertheless experienced a few vertiginous attacks, commencing with tinnitus aurium, before being completely well. He took during two months from one and a half to two drachms of bromide every day, without any cutaneous eruption. Towards the end of September the use of strong coffee could not counteract the drowsiness, and the patient displayed every sign of bromism. The salt, of course, was discontinued for two days before resuming its exhibition in doses of thirty grains, and afterwards decreased to twenty grains three times daily. The amount of bromide of potassium was carried up to thirty grains, whenever the patient dreaded, through his nervous condition, the approach of an attack. The conium was steadily employed in repeated large doses of from six to eight grains, with half the amount of ergotine. Gymnastics were besides added to the tonic regimen, and medical treatment was suspended in the spring of 1866. No attack of epilepsy has returned up to the end of September of 1869, and the patient considers himself completely cured.

Case XXIII. *Epilepsy.—Amenorrhœa.—Hypodermic injections of strychnia.—Arrest of the fits.*

Miss M—— W——, native of New York, aged 18, of pale, unhealthy aspect. No hereditary taint of nervous disease in her family. Menses appeared at the age of 13; continued irregular in their return, attended with severe headache, until they were completely suppressed, after flowing for some months sparingly, at the age of 16, in February, 1861. The 12th of January, 1861, corresponding to the menstrual period, she was seized with the first epileptic fit. She previously experienced severe headache, with pain in the back, complained of her stomach, and went to sleep in the afternoon, when the spasms occurred. She bit her tongue in this and in every subsequent fit, frothed at the mouth, and continued sleeping after the paroxysm, of which she remained wholly unconscious. The following month, as she expected the catamenia,

another fit seized her suddenly in the daytime, and preluded by the symptoms just described. She was much convulsed, for a long while comatose after the fit, and recovered from it with violent headache. Similar fits, mostly nocturnal, or occurring during sleep in the daytime, have frequently and invariably made their appearance towards that time of the month corresponding to the period of the suppressed catamenia. Different courses of treatment have been unsuccessful to restore the menses, and to cure the epileptic paroxysms.

I saw this girl March 18, 1863. She had gone without fits nearly four months, until the day before she consulted me, when she had an attack in the night. The parents have avoided letting her know the true nature of her complaint, and she believes that she suffers only from amenorrhœa and headache. She constantly complains of distress in the lower part of the back, extending to the hypochondria and downwards to the thighs, with a constant icy coldness of the feet. The depression and want of energy to engage herself at anything are insurmountable; she is very sensitive, and cries on the most trifling remark addressed to her. The appetite is very poor. Tongue slightly coated. Bowels habitually constipated. Pulse 98, small. Respiration 15. Heart's action quick, with a distinct chlorotic humming extending to both carotids. She very reluctantly consented to an uterine examination, which showed the neck pale, prominent, and smooth; external orifice plugged by transparent thick mucus; great tenderness detected on both ovarian regions, and specially more on the left. The uterus slightly anteflexed; did not seem otherwise out of proportion; a fine sound easily penetrated into its cavity. No leucorrhœa had ever existed. Chloro-anæmia had made considerable progress on this girl, and she was therefore advised the following plan:—Cold sponging of the body, and friction every morning immediately after getting up; to take at bedtime an alkaline tepid sitz-bath for about twenty minutes; a generous diet, with claret or Burgundy wine at meals; exercise in the open air, and with the dumb-bells; to take three times daily the following mixture:—

℞ Ammon. acetat., f. ʒ j.
Acidi acetici dil., ♏xx.
Tinct. ferri sesquichlor., ♏x.
Aquæ dest., f. ℥ j.
Misce.

and to have at bedtime one tablespoonful of this mixture :—

℞ Potassi bromidi, ʒ iv.
Tinct. sumbul, f. ℥ iss.
Aquæ pimentæ, f ℥ ivss.
Misce.

To maintain counter-irritation over the ovarian regions, by painting the parts with blistering collodion, and finally to move the bowels when necessary, by an enema of tincture of assafœtida and turpentine.

This plan, with unimportant variations, was observed. April passed without any fit, nor much headache. The pain in the hypochondria and back mitigated considerably, with the sitz-baths and counter-irritation to the ovaries; but the girl complained, as ever before, of coldness in her feet, and to prevent it one-ninetieth of a grain of strychnia was injected in each leg, renewing the operation every other day, and the coldness completely disappeared after eight injections.

The dose of bromide of potassium was increased to ʒ ss.—to be still taken at bedtime. Improvement continued, but the 9th of May, at 2 o'clock in the morning, she had an epileptic fit less severe than the former ones, and preceded by headache during the evening. She exhibited the next morning scattered petechiæ in the forehead, which had been also noticed after the previous attacks. She further appeared languid and hysterical, crying a great deal upon awaking in the morning and during my visit. Ordered: An alkaline bath, and a stimulant mixture with valerianate of ammonia, tincture of sumbul, and cinnamon water. The 6th of June the dose of bromide of potassium was augmented to forty grains, to be taken in the morning and evening. June 16th she was fully under the influence of the bromide, staring fixedly at persons around her in a sort of ecstatic condition, with pupils hardly contracting and much dilated, conjunctivæ injected, cheek and ears flushed, and occasionally, as the mother states, under the delusion that she hears her parents talking to her when they do not. She is very sleepy most of the time. She complains of no pain. Pulse 65, regular, rather feeble. Respiration 16. Tongue with a thick white coat, increased secretion of viscid saliva, offensive breath and congestion of the pharynx. Appetite lost, bowels regular, rather loose. Cutaneous eruption quite thick in the face and neck. Although these effects of the bromide of potassium were explained in anticipation to the mother, she became very uneasy about the state of her daughter. A tepid bath, strong coffee, and the above stimulant mixture dissipated all the symptoms upon discontinuance of the bromide.

June 21st she had a slight flow of blood after the tepid alkaline bath in the evening, but the discharge did not seem much favored by further continuance of warm applications to the thighs and lower region of the spine. The girl awoke the next morning very depressed and hysterical. She remained in bed, went to sleep in the afternoon, and on awaking was seized with general tremor and a very strange feeling, with twitching of the head and restlessness, ending in a fit of crying, after an enema with one ounce of tincture of assafœtida and half an ounce of spirits of turpentine, mixed with eight ounces of mucilage.

A few days after this attack she was taken to Sharon Springs. She was advised to continue using at bedtime, only on the approach of her menses, a mixture with valerianate of ammonia, and twenty grains of bromide of potassium. She had kept to that time pretty regularly with the chalybeate mixture ordered from the beginning. Menstruation recurred in July, scanty, attended with a languid hysterical condition and without any convulsive paroxysms. On her return to the city, September 8th, she appeared very much improved. She had just passed through her menstrual period, which lasted three days, with comparatively little disturbance. The treatment was not materially modified thereafter, and together with a nutritious diet, and gymnastics, the girl recuperated her strength and regained the regularity of the menstrual function, no more epileptic attack having recurred to this date—January 4, 1870.

The benefit that may be derived in amenorrhœa, from remedies mainly directed to the ovaries, is manifest in the preceding as much as in other similar examples here narrated. I deem it unnecessary to repeat what I stated on this subject when speaking of the accidental causes of epilepsy.* I then also dwelt

* I take occasion to remark that, after these pages had gone to press, a female epileptic died at the Hospital with all the symptoms of spinal apoplexy, confirmed by the autopsy. She had been subject to deranged menstruation. During her last days the right parotid and sub-maxillary glands became enlarged, sympathetically, on inflammation of the right ovary, as manifested by the autopsy. This is the first anatomo-pathological confirmation of this sympathy of the ovaries, which I pointed out clinically some time ago.

on intemperance as one of the commonest sources of the spasmodic paroxysms, and of which Case XXVI. is an illustration. What I should particularly notice in reference to the two last instances is: the advantageous employment of hypodermic injections of strychnine to promote a regular capillary circulation, and thus remove the uncomfortable chilliness of the extremities in epilepsy. This means has been resorted to with some of the cases that have found a place here, and I consider it a most valuable adjuvant to a successful treatment of epilepsy. In 1859, Dr. Behier read before the Imperial Academy of Medicine, in Paris, a paper on the use of medicinal hypodermic injections for the treatment of neuralgia and other nervous affections. Reference was made in this interesting communication to the successful injection of sulphate of strychnia in seven cases of paralysis, of which four were cured and the remainder unimproved. Professor Courty, of Montpellier, published in 1863 three cases of facial palsy and one of paraplegia of a year's standing, which readily yielded to a few subcutaneous injections of strychnia. Guided by the successful results of the French physicians just cited, I began to study the effects of strychnia hypodermically administered to some of my paralytic patients. In April, 1869, Mr. Charles Hunter, of London, contributed to the British and Foreign Medico-Chirurgical Review an interesting communication "On Strychnia Hypodermically Administered in Paralytic Affections," and a month later, May 28th, 1868, I read before the Con-

vention of the Connecticut Medical Society, at New Haven, a paper, with an abstract of the most striking examples of paralysis I had cured with hypodermic injections of strychnia during the previous five years, and the effects usually attending said injections. I also incidentally alluded in that communication to this hypodermic treatment in other diseases than paralysis, and especially in epilepsy. I should add, that since that paper was read I have had occasion to try three times, in unequivocal instances of myelitis, the hypodermic injections of strychnine, with the beneficial results reported by Hunter.[1] There is now a patient at the Hospital, who came in with myelitis in its most advanced stage, attended with cystitis, complete incontinence and alkalinity of urine, abdominal constriction, paraplegia with anæsthesia, cramps and jerkings of the lower limbs, bed-sores over the sacrum, etc., and who has exhibited great amendment of his symptoms, with power to retain his urine,—no longer alkaline,—partial return of sensibility, and healing of the bed-sores over sacrum, upon the daily hypodermic injection of one-fiftieth of a grain of strychnine, continued to this date for about one month. I am not aware of the hypodermic employment of strychnine in epilepsy, or in any of those nervous diseases where the peripheral circulation becomes deficient, causing chilliness and altered nutrition of the skin in the limbs. I have noted these phenomena in many of the foregoing cases, and I may here assert, upon extensive observation, that the subcutaneous injection of strychnine is most efficient

to produce a permanent warmth and to remove the coldness of the extremities which troubles the epileptics so much; while it also increases nutrition, contributing to cure local cutaneous eruptions, rendering more active the depressed capillary circulation. Strychnine under such circumstances acts unquestionably very different from what it does when absorbed by the digestive organs. Anstie says: "I am convinced that there is something which we do not understand, and which is completely distinct from the familiar effects of this drug (strychnia) in its action when given in very small quantities. I am by no means sure that the effects do not vary, perhaps considerably, according to the taker's constitution; but this at any rate I believe to be a nearly universal rule, that doses which fall short of producing any poisonous effect increase the activity of the systematic circulation, diffuse a comfortable feeling of permanent warmth over the body, and favor the progress of local nutrition when this is deficient." * The truth of these remarks applies more forcibly to minute doses of strychnia hypodermically thrown into the system, which act directly on the vaso-motor nerves. Strychnine, even when administered by the mouth in fractional quantities, is apt to exalt quite readily the excitability of the spinal cord. That epileptics, as pointed out by Marshall Hall, may improve when using strychnine is an indubitable fact, although it is not easy to limit the quantity of strych

* Stimulants and Narcotics, p. 143.

nia, or the length of time that they should take it without risk of aggravating instead of preventing their attacks. A small dose, repeated for several days without mischief, may suddenly originate violent outbreaks of epilepsy. Never have I met with this effect upon prolonged continuance of the injections, although it is true that I have very seldom gone beyond one-sixtieth of a grain of strychnine to stop chilliness and cold in the limbs of epileptics. The effects of one-ninetieth of a grain, which I ordinarily inject, may last for some hours or even for a day. The rapidity with which the action of the drug is manifested is strikingly illustrated in a female at the Hospital, whose feet were very livid, cold, and painful. Dr. W. H. Bennett, Assistant Physician, injected one-sixtieth of a grain of strychnia in the legs, with immediate relief of pain and increased activity of the circulation, with natural warmth. I have not observed inflammation, or suppuration after the punctures, but on the contrary the injections have brought about rapid healing of atonic ulcers or eruptions in the limbs of inveterate epileptics and paralytics. Generally, I insert the needle of the syringe deep into the skin, and draw out part of it before injecting the solution, to avoid throwing it directly into any blood-vessel. The injection should be practised very slowly. A solution of pure strychnine, with one-hundredth or a smaller fraction of a grain to the drop of distilled water, allows us to carry the local action of strychnine at the same time into more than one limb.

I described in Case XVI. the effects of woorara,

injected hypodermically, in epilepsy. I have had the opportunity of trying this agent recently on three more epileptics: the woorara was kindly given to me by Mr. J. Frey, Chief Druggist to Bellevue and the Blackwell's Island Hospitals. The solution I employed contained one grain of woorara to ten drops of distilled water, and I previously satisfied myself that the woorara exhibited its poisonous properties. The following is an abstract of the results in the seven cases to be added to the one formerly detailed:—

Case XXIV. Boy, aged four years and a half. Fits of petit mal, repeatedly occurring through the day, and less frequent grand mal, with palsy and contraction of right arm. Sixteen injections of woorara, made along the cervical region, beginning with one-fifth of a grain until reaching half a grain of woorara. The injections were practised almost every day. Effects: Flushed appearance with increased temperature of the face and neck. Alternate contraction and dilatation of the pupils, heavy expression of the eye, with perceptible squinting after the four last injections. Languidity of the child with relaxation of the muscles of the arm. The pulse increased from 92 to 102 and 110; respiration from 18 to 26 or 30. The neck became sore at the site of every injection, and on the six last occasions the woorara was introduced into the traject of a seton inserted in the lower part of the child's neck. No diminution was perceived in the fits of petit mal, nor in the convulsive spasms. This child could be thrown into a fit of petit mal by pressing the spine at the region of the seventh cervical vertebral process, and the phenomenon would be equally produced while he was fully under the influence of woorara.

Case XXV. Girl, aged 18. Had epileptic paroxysms every morning, soon after getting up. Ten hypodermic injections during 13 days, commencing with one-fifth of a grain until injecting one grain of woorara on each of the three last times. No change whatever produced on the frequency of the attacks. The neck became very tender, but none of the punctures suppurated. The girl complained of great dizziness, vertigo, and headache, with numbness and weakness of her limbs after the last injections, when the pulse would increase from 82 to 104, with accelerated respiration, and a general feeling of heat and suffocation. The face would

be congested, moistened by perspiration, the conjunctivæ injected, the pupils dilated and now and then contracting almost to a point, to remain again enlarged for a long while, very slightly sensitive to light. The throat and mouth were dry and parched, and the internal feelings of the patient so unpleasant that she refused to submit herself to further trial. The phenomena just enumerated appeared only after the three last injections.

Case XXVI. Man, aged 38. Having two or three fits every day. Eleven hypodermic injections during two weeks. Inflammation and tenderness, but no suppuration, in the cervical region where the woorara was injected. Pulse raised from 78 to 90 or 96, after injecting four-fifths of a grain of woorara. Same flushed appearance and drowsy expression of the face noticed with the above patient. Dilatation of the pupils, drooping of the eyelids, dimness of sight, persisting five and six hours after the injection; dizziness and loss of power in the limbs, with incapacity to act. The fits disappeared the third and fourth days after the injections, to recur with more severity. The patient even thought that they would leave him more exhausted than before using woorara, and therefore objected to it. He began with three-fifths of a grain, and had on the two last occasions one grain and a fifth of woorara injected in the neck.

Case XXVII. Girl, aged 5 years, with spasmodic fits from three to five times every day. In this instance three injections only were tried. The child would become very much frightened at the operation and would violently resist it. One-fifth of a grain was the dose employed on each occasion, but the excitement of the child did not allow me to recognize any of the effects that woorara might have produced on her.

The three following cases are those observed since October, 1869, and it is unnecessary to remark that in them, as in those just alluded to, all other treatment was discontinued while the woorara was administered:—

Case XXVIII. Female, aged 22. Petit mal and spasms, the latter of a nocturnal character, but both occurring every day. Urine acid, no sugar. Eight consecutive injections in the same number of evenings, beginning with two-fifths of a grain until reaching one grain and a half of woorara, by successively increasing the dose of one-fifth of a grain, and using the highest dose the three last evenings. Pulse 86. Respiration

15 before the injections; after the injections the pulse would be as high as 112; respiration 20. Dilatation of the pupils, congested face, numbness of the head and neck; vertigo. Such were the main phenomena before injecting doses as large as a grain of woorara. The effects were more pronounced upon the injections of one grain or one grain and a half of woorara, when the patient would experience extreme lassitude, with hot skin, congested face, pulse from 104 to 112, accelerated respiration, dimness of sight, and manifest deviation of the eyes. The secretion of urine increased from the beginning, but sugar was detected in it only after the third hypodermic injection of four-fifths of woorara. No change whatever in the epileptic attacks, and the neck became so very tender and stiff, the dimness of sight and weakness so uncomfortable to the patient, that the treatment was discontinued. In this case a small boil originated at the site of one of the punctures.

Case XXIX. Male, aged 18. Spasmodic paroxysms occurring every day, and attended with dementia. Pulse 84. Respiration 14. Urine acid, no sugar. Six injections along the neck, two in the left arm; beginning with three-fifths of a grain until one grain and three-fifths of woorara were used for the injections in the arm. The urine turned diabetic upon the second injection of half a grain of woorara, but the secretion seemed to increase from the commencement, according to the attendant's statement. The fits did not change in the least, whereas the patient appeared evidently unfavorably influenced from the fifth injection of woorara. His face, ears, and neck would become very hot, perspiring slightly; the pupils largely dilated, and sluggish to contract to a strong light; constant closing of the eyelids. The patient would frequently shake, or gape; his respiration would be irregular and sobbing. He would sit indifferently, without moving, in a drowsy state, yet conscious of everything, and, if requested to walk, he did not seem capable of moving his limbs, and would say all the time "I can't see."—"Give me some water." His mouth and tongue were quite dry, but the secretion of saliva and tears was increased after the two last injections with one grain and three-fifths of woorara. The foregoing condition was, of course, observed subsequently to the administration of a dose of a grain or more of woorara, and did not disappear completely until two days after the last subcutaneous injection of one grain and three-fifths of woorara. The neck and arm were red and swollen, but did not suppurate at the site of the punctures.

Case XXX. In this last instance, a girl aged 26 had attacks of

epilepsy, regularly seizing her almost every night. I only tried four injections with her, respectively with two-fifths, three-fifths, and the two last with half a grain of woorara. The urine acid, without sugar, increased from the first injection; but evidences of sugar only appeared after the fourth injection. The patient remained, upon injection of half a grain of woorara, for four hours in a true febrile state, with pupils dilated, great inclination to keep the eyes closed, and incapable of using her limbs. She further complained of dimness of sight, of thirst, and of violent abdominal pain, followed by diarrhœa. Her pulse, from 86 went up to 114, and the respiration from 16 to 28. These phenomena lasted from six to eight hours after the two last injections, and the fits recurred as before every night.

I have previously referred to the interesting researches of Voisin and Louville on woorara. Thanks to their study, I have been able to try woorara in the three last reported cases, with more completeness than I did before. The treatment has not been prolonged in any of my cases as much as in the twelve patients kept under observation by Voisin and Louville; nor did I ever reach the extreme dose of 18 centigrammes, three and three-fifth grains, employed by the French physicians. The largest quantity I injected has been one grain and a half. The phenomena I observed agree with those so thoroughly described by Voisin and Louville. I may further add that, like them, I did not detect any variation whatever from the normal aspect in the condition of the optic disc or the retina, on examining with the ophthalmoscope the eyes of the three last patients submitted to the hypodermic injection of woorara. As to the pulse, I could also ascertain with said patients, that it had not only increased in frequency, but

that the sphygmographic tracings showed a distinct dicrotism in the pulsation—a fact pointed out besides by Voisin and Louville. So far as my own observation goes, I believe that if woorara were of any advantage in the treatment of epilepsy it would have been manifest, at least in preventing the paroxysms in some of my eight patients subject to daily fits, and fully brought under the physiological action of woorara. I despair, therefore, of its usefulness, and have desisted from further trial with the substance in this direction. This conclusion is in no way discountenanced by that arrived at by Voisin and Louville, who, after employing woorara hypodermically for several months in twelve epileptics, state: "As regards the definite results upon the use of woorara in epilepsy, we cannot at present affirm anything in any way. We have not seen that it produced harm; but nothing authorizes us to praise its efficacy."* Such results are not either incompatible with those so favorably obtained by Thiercelin, if we bear in mind that this experimenter applied the woorara over blisters in the cervical region, and that the blisters by themselves are powerful means to check the epileptic paroxysms.

Narcotics are usually worse than useless in epilepsy, as asserted by Duckworth Williams. Morphine may arrest the repetition of fits; but as soon as its effects subside, the paroxysms return with their former, if

* Journal de l'Anatomie. Tome IV., p. 134.

not with greater intensity. This harm has been again and again observed with patients at the Hospital; and I hear it often acknowledged by those who resort to the unfettered use of morphine to avoid renewal of the spasms, or their subsequent agitation. There is, nevertheless, a stage after a series of fits, when epileptics remain devoid of all energy, and very low, in which morphine, cautiously administered in doses not exceeding one-fifth or one-fourth of a grain, acts as a cerebral tonic, inducing quietude and sleep. Females seem more prone than males to run into this depressed condition, and to tolerate morphine better, because, perhaps, as shown by Trousseau, they generally stand much higher doses of morphine than males. However, I have seen much surer good accrue from the subcutaneous injection of one-sixtieth or one-fortieth of a grain of atropine, to relieve the above depressed condition. After it, the pulse has gained regularity and force, with rapid and persistent disappearance of the asthenic symptoms. I commonly combine atropine and morphine to lessen pain from blisters, or cauteries to the spine and other parts of the body in epilepsy. I am at present attending a young lady with spasmodic fits, preceded always by an aura starting from the toes in the left foot, which are strongly cramped for a few seconds before the irritation reaches the nervous centres. A ligature around the leg fails to stop the aura, which is, however, prevented constantly by stretching the toes forcibly when seized with the cramps, and thereupon the fit avoided. This

patient has objected to cauterization of the foot, and I have injected subcutaneously every night one-sixth of a grain of morphine with one-fortieth of a grain of atropine. The fits, which occurred almost daily, disappeared with this treatment. To satisfy myself that the aura was held in check by the injections, I discontinued them five days, and the fits recurred with the primitive frequency, and initial cramp in the toes. I have, of course, resumed the hypodermic injections to the left foot, but cannot boast of anything beyond the arrest of the aura, expecting that removal of the peripheral irritation will finally bring about a cure of the epileptic fits.

I know of no safer narcotic in epilepsy than conium. I prefer it to belladonna. This latter has appeared to me more active with children than with adults, and its value palpable mainly in vertiginous epilepsy, or when the two forms of epileptic attacks are combined. Otherwise I have pushed belladonna to the highest possible doses in ordinary convulsive fits, without in any way obtaining any reliable results. Not so, however, with conium, which is of great assistance chiefly in epilepsy attended by cerebral derangement or vertigo. But conium, to act favorably, should be administered in large doses, from four to six or eight grains of the extract, or from two to four or five drachms of the succus, taken two or three times a day. The fluid extract of the American Pharmacopœia is often inert, not always of the same strength, and weaker than the succus extracted from fresh

leaves of hemlock. I prescribe either preparation, commencing with half a fluid drachm, and gradually increase the dose, according to the operative effects obtained. As established by Harley,* whose researches have guided me, and as I have controlled it, conium diminishes the irritability of the spinal cord, without distinct interference with the sensory functions, and has a special narcotic influence on the pneumogastric nerve. It tranquillizes and renovates the whole muscular system, hence its great usefulness in epileptic insanity. Ordinarily, I associate conium to ergotine, which appears to help the sedative effect on the capillary circulation of the nervous centres.

I might assert, in reference to pain, that I do not recollect any instance where its severity upon traumatic injury, or accidental extensive burns, or even upon surgical operations, or during labor, has originated a fit in any of the epileptics under my observation. It is when the patient has no pain, or during sleep, that the convulsive paroxysm breaks out, and if I were to conclude from the numerous instances I allude to, I should consider painful excitations and epileptic fits antagonistic to each other. I never dread the occurrence of a fit in cases of serious traumatic injury while the patient is in pain. Moreover, I have noticed epileptic maniacs who, in the interparoxysmal period, tore off the dressings of their wounds, or inflicted on themselves fearful injury, without the least

* Med. Times and Gazette, March 21, 1868, p. 325.

sign of pain, because they actually exhibited, as I have ascertained, complete anæsthesia or analgesia of the skin. They, on the contrary, complained of soreness in their wounds as soon as they recovered from the shock to the will and inward feeling caused by the epileptic fit, which seems to blunt no less the perception of pain.

In 1857, Sieveking read before the Medical and Chirurgical Society of London a paper on epilepsy, which called forth a remark on the part of Sir Charles Locock, who stated as the result of his experience, that out of fourteen or fifteen cases of hysterical epilepsy treated by bromide of potassium, only one-half had failed to improve. Brought out under such high auspices, the bromide of potassium soon began to supplant the other remedies, and to be extensively used in epilepsy. It was not long, however, before the statements in its favor, by MacDonnell, Brown-Séquard, Radcliff, etc., were contradicted, particularly by those who failed to obtain the successful results advocated chiefly by English experimenters. More than twenty years ago, Puche described the physiological action of bromidè of potassium, exhibited in large doses—four and five drachms a day—and it is curious to see how generally ignored are Puche's researches, embodied in the inaugural theses of his pupils, Drs. Huette and Rames. Gubler and Debout had occasion to confirm thereafter the facts advanced by Puche, and Debout particularly insisted on the hypnotic virtues of the bromide of potassium. The

drowsiness, stupor, and hypnotic effects, the trouble of sight and hearing, the hypersecretion of saliva and congestion of the throat, the delirium, unsteady gait, and weakness of the limbs, the anæsthesia of the skin and mucous membranes, the anaphrodisiac properties, were all pointed out by Puche, as the physiological results of large doses of bromide of potassium. In 1865, Professor R. Bartholow published some interesting experimental investigations on the action and uses of the bromide of potassium in the *Cincinnati Lancet and Observer*. The physiological effects of bromide of potassium, due to its prolonged administration, according to Professor Bartholow, may be summed up as follows: 1st. It diminishes and ultimately entirely neutralizes the sexual appetite; 2d. It produces weakness of the muscular system; 3d. It is irritant to the stomach if given in considerable doses; and 4th. It interferes with the secondary assimilation, lessening the retrograde assimilation of tissue. Professor Bartholow does not allude to the appearance of the skin and mucous membranes, which is so conspicuous with bromism, or upon prolonged administration of the bromide of potassium. Eulemberg and Guttman, in 1867,* established, as the result of a series of experiments, that bromide of potassium exerts a paralyzing influence upon the heart and central nervous system, and the poisonous effects are due to the potassium and not to the bromine. According

* Gazette des Hôpitaux, No. 77, 1867.

to [Laborde,* bromide of potassium is not a cardiac poison, nor does it paralyze the muscles, or the encephalon; its primary action is exerted upon the spinal cord, determining essentially an impairment or destruction of the property which it possesses of presiding over the reflex phenomena. Laborde does not attribute the action of the salt to the potassium itself. I am not prepared to decide whether the bromide of potassium operates essentially on the heart and central nervous system, or on the spinal cord alone, although I feel disposed to admit—that it operates actively in a paralyzing manner on the excito-motor power of the spinal system and vaso-motor nerves, as advanced by A. Voisin † and Martin Damourette.‡ Bromide of potassium prevents structural changes by opposing irritation of sympathetic nerves, which is a very capital element of derangement of nutrition. I am also satisfied, as I have previously shown, that the quantity of urea in the urine is notably lessened under the use of bromide of potassium; but the secreting activity of the kidneys is on the contrary increased. The diminution of urea has been already noticed also by Professor Bartholow. The elimination of the bromide through the kidneys takes place immediately upon its exhibition, and persists three and five days after its discontinuance. We have detected evidence of bromine in urine tested a week after the patient

* Gazette des Hôpitaux, No. 83, 1867.

† Bulletin Général de Thérapeutique, 15 et 20 Août, 1866.

‡ Gazette des Hôpitaux, 29 Fév., 1868.

had ceased the use of the bromide; a fact also established by the researches of Dr. G. Namias.* I have not been able to ascertain precisely the conditions favoring the elimination of the bromine by the kidneys; but its proportion has been found increased in the urine secreted after meals, or any prolonged gymnastic exercise. Evidence of the bromine has been manifest in the saliva of those taking the remedy. I cannot say that gastric irritation supervened on any of the cases where I have employed large doses of bromide of potassium. "Among seventy patients who were taking enormous doses of bromide of potassium, Huette met with gastritis and diarrhœa only five times." † One or two patients out of twenty-four, examined by A. Voisin,‡ exhibited diarrhœa, dyspepsia, or slight icterus. Diarrhœa is recorded but three times among 136 patients, who have been using regularly for a year or longer period, from twenty to thirty grains of bromide of potassium thrice daily. In one female the salt in drachm doses would induce nausea and purging; and in an epileptic soldier at the United States Central Park Hospital, fifteen or twenty grains of bromide of potassium would always produce vomiting. He became epileptic upon gunshot wound of the skull. It has been advanced by some writers that

* Académie des Sciences de Paris, 20 Mai, 1867; and Giornale Veneto di Scienze Mediche, Tome XII., p. 371.

† Traité de Thérapeutique, par Trousseau et Pidoux. Tome I., Paris, 1862, p. 334.

‡ Loc. cit.

pulmonary tuberculosis is hastened in epileptics by the bromide of potassium, but I have not seen it. Williams * says that an epileptic girl soon died with tubercles of the lungs after administration of the bromide, and out of truth Williams is compelled to confess that he doubts whether the bromide of potassium had not something to do with this poor girl's death. I have most closely investigated the relations of pulmonary tuberculosis and epilepsy, and undoubtedly the genesis of tubercles in the lungs is favored by the lesion in the medulla oblongata proper to epilepsy. I have traced the pulmonary trouble from its inception, and feel convinced that the association is more than a casual coincidence of both morbid conditions. I have been no less struck with the frequency of tubercles, or other pulmonary lesions, I have met with on post-mortem examination of epileptics. The following is a very interesting instance of the kind :—

Case XXXI. *Epilepsy. Fibrous tumor pressing the left olivary body and pneumogastric nerve. Tuberculosis of the lungs.*

S—— Y——, aged 40, single, entered the Hospital April 22d, 1869. Her mother died phthisic. She had fits in infancy, which never repeated until November, 1868. They had not been frequent, but she was subject to vertigo, after which she would feel extremely sleepy and would hardly be roused, remaining with indistinct articulation. Had amenorrhœa and pain in the back and chest. Perspired considerably at night, with clear symptoms of phthisis. She was sometimes cataleptic after the fits, occurring at distant intervals, but in great number. One day last May she had as many as 200 fits, and on the two next occasions, in November and

* On the Efficacy of the Bromide of Potassium in Epilepsy and Certain Psychical Affections, p. 11.

December, she had, respectively, 108 and 79 paroxysms. She took to her bed in a state of great depression, and without any fits, she died March 25th, 1870. A tumor was suspected, though I could not precise its location, in the vicinity of the medulla oblongata.

Autopsy. Encephalon weighs 41 ounces, brain proper 29 ounces, right hemisphere 14¾ ounces, left hemisphere 14¼ ounces. Cerebral tissue congested throughout; gray matter thin in some of the convolutions, with a rosy hue, and easily detaching from the meninges. A fibrous tumor, the size of a pea, continuous with the spinal dura mater, presses on the left corpus olivarium. The left pneumogastric is compressed and a deep sulcus is caused by the tumor on the medulla. The right lung is intensely congested at the base and emphysematous at the apex. The left upper lobe, carnified, is stuffed with tubercles, part of which, about one inch in diameter, have suppurated. Both lungs were crowded very high up, the diaphragm reaching above the level of the nipple. Nothing remarkable with the heart. Liver apparently enlarged; weighed, however, only 41 ounces. The left lobe extended far to the left, and the left lobe was excessively congested. Spleen of very dark appearance and very soft. Transverse colon descended and lay on the pubic bones, well filled with feces. Ilium congested and contracted. Kidneys highly congested, the right one containing a milky fluid. Uterus with a large fibroid tumor in the posterior wall, and with numerous minute transparent cysts in the peritonæum over the organ and its appendages.

Finally, the medulla oblongata exhibited peripheral sclerosis of the left olivary body at the site of the depression; the restiform bodies were slightly involved also; the inferior surface near the middle line of the pons Varolii exhibited small discrete patches of sclerosis; and the left pneumogastric nerve was distinctly implicated. The relation between the injury sustained by the pneumogastric and medulla and the left lung is self-evident to need any further remarks on the subject.

By adding 137 patients, who have been under my direction since the foregoing synoptic tables were made out, we will have a total of 443 epileptics: 207 males and 236 females. Of them 27 never used bromide of potassium, which proved powerless in 98 cases—55 males and 43 females. Among the males, two dis-

played a hereditary taint of nervous disease, five were epileptic since infancy, three demented, and six of the remaining had epileptic insanity, the fits outbreaking in two of them after injury to the head. Five females acknowledged a hereditary predisposition to epilepsy, seven had fits since infancy, whereas twenty-six of the remaining exhibited insanity, four of them being demented. None of the 318 patients who have regularly used bromide of potassium has taken less amount than twenty grains thrice daily, or for shorter period than seventeen weeks. The maximum doses ranged from forty to seventy-five grains, kept up every three or four hours until inducing bromism. There is at the Hospital a girl who took, for twenty-one days, three drachms and a half of bromide of potassium daily without any sign whatever of intoxication and with great improvement. This, however, is an exceptional instance, since seldom have I seen forty grains, repeated thrice daily for five or six days, without determining bromism. The bromide has been prescribed usually in a mixture with the fluid extract or succus of conium, or with the tincture of sumbul, of calumbo, of cardamom, etc., and five or eight minims of Fowler's solution. In syphilitic epilepsy, or when the fits were originated or aggravated by alcoholismus, or again, whenever signs of active cerebral degeneration were apparent, the iodide of potassium has been combined with the bromide. The bromide of ammonium and the carbonate of soda have been less frequently administered, because I have not recognized any mark-

ed adjuvant effect from their admixture, which renders more impalatable to the patient the already disagreeable taste of the solution of bromide of potassium. The bromide mixture has been given in the daytime, fifteen or twenty minutes before meals; patients have been directed to drink strong coffee with their meals, or through the day when using large doses of bromide of potassium. The operation of the remedy seems aided by this practice, and the supervention of bromism very materially delayed, epileptics being thus able to take nearly twice as much bromide without necessity for suspending it, which is important to secure the arrest of the disease. As to ammonia, I prefer prescribing it, when needed, in any simple solution,—the aromatic spirits, or the carbonate, the valerianate, the acetate, the muriate of ammonium. The acetate of ammonium associated with the sesquichloride of iron forms, perhaps, one of the best chalybeate preparations I have employed in epilepsy. I prescribe it according to a formula borrowed from Basham's excellent work On Dropsy Connected with Disease of the Kidneys.

℞	Ammon. Acetati	f. ʒ j
	Acidi acetici dil.	♏ xx
	Tinct. Ferri sesquichlor.	♏ x
	Aquæ	f. ℥ j.

Misce ft. haustus.—To be taken three times daily.

In many cases where improvement accrued from the administration of the bromide of potassium, oxide of zinc, gr. v. to gr. xxx., or extract of belladonna, gr. j.

to gr. ij. taken daily, had been previously resorted to without any good. In those relieved by full doses of bromide of potassium, forty, fifty, sixty, or seventy-five grains have been administered every third or fourth hour, occasionally, until producing bromism. The symptoms then exhibited have been: congestion with swelling of the fauces and of the tongue—redness of the conjunctivæ and cheeks; dilatation of the pupils, dimness of sight, thickness of speech; slowness of pulse and respiration; increased secretion of the salivary glands and kidneys; in some instances hallucinations of sight and hearing, and mania—on three occasions of a suicidal nature. The swelling of the fauces, extending to the Eustachian tube, has determined deafness in some cases, but readily disappearing upon discontinuance of the bromide. The anæsthesia of the skin and mucous membranes has been very conspicuous at this stage of intoxication, as also a tottering gait, with inability to steady exertion of any kind (writing, buttoning up the clothes, etc.), and an overwhelming drowsiness. In no case has the appetite to eat or to smoke been completely lost. I have long ago noticed that fetidity of breath, usually an accompaniment of the exhibition of bromide of potassium, occurs earlier and more remarkably in those who do not attend to regular cleanliness of the teeth; hence its more striking character among hospital patients. Its occurrence is of transient duration; ordinarily ceasing upon continuance of the bromide. The phenomenon is not solely due to the elimination of the salt through

the salivary glands; for frequently a test of the saliva will exhibit the bromine without any existing offensive smell of the breath. On no occasion, however, have I met with this latter without more or less swelling of the pharynx and tongue. The hypersecretion of the salivary glands has been preceded not uncommonly by dryness of the mouth and fauces. The perspiration has become offensive, in some instances, upon employment of even small doses of the bromide. The skin has displayed a peculiar brown hue, more striking in the forehead and neck, and, more generally, a papular eruption upon exhibition either of small or large doses of bromide, and irrespective of any condition of bromism. I discovered, four years ago, that the association of the bromide of potassium and the arseniate of potash avoids the eruption just mentioned. We, however, fall short of this result if alkaline baths are not employed in conjunction, or if the eruption be not previously arrested on discontinuance of the bromide. From five to eight minims of Fowler's solution, added to each dose of the mixture of bromide, will prevent the cutaneous eruption. The papules may break out in confluent form in the dorsal region of the hands, the elbows, the knees, or legs, causing suppuration, which I have met with sometimes throughout an extensive surface of the subcutaneous tissue. Females seem more liable to the eruption, while children remain freer from it.

I may say with Duckworth Williams, and resting on my own observation, "that neither the male nor

the female masturbator is improved in his or her habits from the use of the bromide of potassium in even the largest doses." Nor am I quite sure that bromide re-establishes the catamenia in amenorrhœa, although menstruation for four months absent reappeared with decreased severity and frequency of the fits in a girl at the Hospital, after taking thrice one drachm of bromide of potassium with five minims of Fowler's solution. Concerning the anaphrodisiac properties of the bromide of potassium cases have been here reported where they were unsuccessfully tried. I have carried the salt up to the highest limits—two and a half drachms in six hours—in a female nymphomaniac and paraplegic, without further effects than those of bromism. The relief has proved no more positive with the epileptics addicted to masturbation. Once the bromide had to be discontinued at night, on account of its inducing lascivious dreams, with seminal emissions, where they did never exist before. Similar phenomena have been observed thrice by A. Voisin, who met with partial satyriasis, nocturnal erections, lusty dreams, and great seminal loss produced by the bromide of potassium. I do not believe that the inward feeling that prompts self-pollution acknowledges its origin essentially in the genital organs. I have seen maniacs in restraint, inventing some way to gratify their morbid sensuality, and, most certainly, it is the thought rather than the material act which contributes to awake the sensual desire dwelling in no determined organ of the body. For these reasons, I

consider unwarranted the claims of anaphrodisiacs generally, and the value of clitoridectomy, or castration, for the relief of epilepsy, very much over-estimated. My opinion is unreservedly against the latter operation, which I would deem justifiable only if there were a manifest source of irritation, or a disease of the testicle, requiring its removal for the cessation of the spasmodic paroxysms. I must, however, remark, that although experience leads me to doubt of the anaphrodisiac virtues of the bromide of potassium, yet I am far from denying that anæsthesia of the mucous membranes, which attends its exhibition, might not prove of great avail in urinary diseases, and chiefly to soothe urethral irritation.

I have watched twice the influence that bromide of potassium might exert on the fœtus. One woman took daily throughout gestation one drachm of bromide in tincture of sumbul, and had not one single fit; another woman was troubled with three attacks during the last two months of pregnancy, and the dose of bromide had to be raised, during a short period, from twenty to thirty grains thrice daily on each of the occasions. The bromide was exhibited with fluid extract of conium and Fowler's solution. In neither case did the fœtus suffer from the above treatment, both children being quite healthy, and respectively three years and one year old. Furthermore, in case 107, Synoptic Table of Female Epileptics, large doses of bromide of potassium were as powerless on the fits as on the course of pregnancy, which was the strange

cause of epilepsy and mania. Epilepsy and pregnancy appear not to influence each other, and seldom are paroxysms induced by labor. In this my experience agrees with that of Laforgue,* but I could not admit with him, that the fits occurring during pregnancy entail no ill effects on the offspring, commonly born alive and in good health. The very case last alluded to contradicts the statement. I could cite other four instances of children begotten by a mother subject to fits throughout pregnancy, and who died in convulsions soon after their birth. Finally, a lady, now under my care, had a succession of fits within a week in the seventh month of pregnancy, and two days after the last attack she miscarried. The fœtus appeared to have died some days before.

It is not rare to observe irritability of temper among the phenomena of bromism. The bromide of potassium, in as low a dose as ten grains, brought on mania with suicidal tendency and hallucination of hearing in one female when she entered the Hospital in October, 1866. She has afterwards taken twenty grains of the salt, thrice daily, favorably, and without the above symptoms. Two other females are seized with suicidal mania when the bromide is carried up to larger doses than thirty grains. Another female, again, becomes maniacal on using twenty grains of the salt, while two of her companions are rendered by the same amount, one melancholic with delusions of hearing,

* Journal de Médecine Mentale, tome ix., p. 129, and Revue Médicale de Toulouse, 1867.

and the other melancholic and drowsy. Mania, once homicidal, has occurred three times among males, who, however, have shown oftener hallucination of sight or hearing upon exhibition of high doses of bromide of potassium.

Large doses of bromide of potassium do not act favorably when suddenly employed in the commencement of the treatment, or without any other adjuvant; but it is necessary to bring epileptics fully under the physiological effects of the bromide, and then suspend and decrease the amount of it for a while, in order to secure beneficial permanent result. Epileptics should not be accustomed to tolerate the bromide of potassium, or exposed to slow intoxication, by constantly using larger quantity than one drachm in twenty-four hours, when the fits are checked, and there are no signs of impaired general nutrition. Nor should the dose be ever augmented, or its exhibition prolonged, unless the condition of the patient indicates the approach of trouble, or unless it be required by the frequency of the attacks. I believe that such fatal issue as that reported by Dr. Hameau* may be avoided by this course. So far as my experience goes, it is exceptionally that patients would stand daily more than one drachm and a half of bromide of potassium without soon exhibiting symptoms of bromism. There are signs indicative of the approach of the bromide intoxication; such as, the enlargement and sluggish contraction of the pupils, drowsiness after eating, and a

* Journal de Médecine de Bordeaux, Mars, 1868.

marked slowness of the pulse. In some instances patients become more irritable, though morose and apathetic if left by themselves. I have never pushed the bromide much longer after these symptoms have appeared.

No difference has been remarked in the effects upon those patients who were almost always attacked during the night, and those who had fits in the daytime. Duckworth Williams gives as the result of his experience "that both amongst the males and the females the decrease in the number of fits has taken place in the day, whilst those during the night were of nearly equal number when the patients were taking medicine as when they were not; and, moreover, it will be found that those patients who were most frequently attacked during the night were those who received least benefit from the use of the medicine."* I need not repeat that I have not based the treatment of epilepsy mainly on the use of the bromide of potassium, which may account for the difference between my results and those of the distinguished English alienist. I here present a table with the number of fits, during 1869, of 33 epileptics, some having already tried bromide of potassium before I saw them, but never in large doses nor until producing bromism. I do not submit them as cured, but to show the benefit derived from the course I pursue, most of them being afflicted with long-standing epilepsy and more or less impairment of the intellectual faculties.

* Op. cit., p. 15.

MALES.													FEMALES.											
Age.	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Age.	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.
21	18	14	19	20	16	21	19	..	..	..	..	..	18	26	7	5	6	4	..	..	..	..	..	..	..
32	26	31	28	12	3	5	1	..	..	1	..	..	26	34	28	22	22	6	3	..	1	..	..	..	..
28	47	35	18	6	8	..	..	..	..	..	..	..	16	8	4	8	..	..	..	..	..	..	..	..	..
14	10	18	11	3	2	4	..	..	1	..	..	..	37	14	2	1	1	4	1	..	..	..	..	..	..
7	39	4	..	..	..	..	..	..	..	..	..	..	35	19	14	14	..	6	..	..	..	..	..	..	..
12	28	10	2	..	..	..	..	..	1	..	..	..	12	40	21	16	7	3	2	3	3	..	3	..	..
9	4	6	..	..	..	..	1	..	..	..	..	..	18	13	12	8	10	..	7	2	1	..	..	1	..
26	8	6	9	2	..	..	..	..	..	..	..	..	22	16	14	14	9	3	6	2	3	..	..	..	..
35	18	16	14	6	2	..	1	..	..	..	..	..	40	35	5	16	4	6	..	..	..	..	..	..	..
30	5	7	2	..	..	..	..	..	..	..	..	..	17	14	3	2	1	..	..	..	..	..	..	..	..
27	13	9	11	3	1	1	..	..	..	..	..	..	23	16	5	4	6	..	..	1	..	..	..	..	..
12	19	15	17	10	8	6	3	..	..	..	..	..	17	4	2	..	..	2	..	..	..	..	..	..	..
10	22	14	20	16	5	2	5	..	..	..	..	..	13	8	3	1	..	..	..	..	..	..	..	..	..
6	28	19	14	9	4	6	3	7	2	..	2	..	19	15	6	2	..	..	1	..	..	1	..	..	..
9	32	29	32	8	16	6	6	4	..	1	..	..	24	28	12	5	1	..	2	..	..	..	..	..	..
22	17	3	1	2	1	2	1	..	..	..	..	..	16	30	19	11	7	2	2	..	1	..	..	..	..
..	..	..	..	..	..	..	..	..	..	..	..	..	19	27	15	9	6	1	1	1	1	..	..	..	..

The last four males and the last seven females had the epileptic attacks always in the night, and they evidently improved under the treatment. I employed counter-irritation to the neck—blisters, setons, or red-hot iron—in six males and in four females. With all but three of the whole cases—two males and one female, I resorted occasionally to subcutaneous injections of strychnine or of atropine. Either the cold shower or tepid alkaline bath, or packing in the wet sheet, constituted a no less prominent part of the treatment, in addition to the use of conium, as previously indicated, and to a good nutritious diet with plenty of coffee. Each of these patients has been kept at different intervals of two or three weeks in a condition of bromism, and continue taking daily from one drachm to one drachm and a half of bromide of potassium.

It is out of my purpose to dwell on the results that may be derived from the various drugs praised for the cure of epilepsy. I have tried in several instances, and repeatedly at the Hospital, cotyledon umbilicus, sulphate and oxide of zinc, oxide of silver, belladonna, atropine and valerianate of atropine, digitalis, etc. None of these substances acts in any specific manner, nor more powerfully than the bromide of potassium on the nervous system. I do not wish to speak too absolutely on the subject, but this is the conclusion to which my own experience has led me. Cod-liver oil ranks ahead of nervine tonics, and it has been freely administered in most of the cases here considered. I look upon it as one of the most valuable remedies, though not prescribed as frequently as it should be in epilepsy. I ought not to forget to mention the valuable remedy I have found in turpentine to remove the irritable state succeeding the fits, whenever they leave behind insanity with propensity to renewed paroxysms. In uterine epilepsy, turpentine, as remarked by Pritchard, proves very useful. I prescribe it ordinarily with tincture of assafœtida in injections, and have obtained much benefit from it. Reliable hopes may be also entertained in the application of electricity, which may afford a real amendment of the epileptic attacks. A low-tension current from six or twelve cells of a galvanic battery, applied daily, for a few minutes, to the spine, has rendered great services in some instances. Although I cannot present a complete cure of epilepsy by this means, it

is nevertheless the fact that I have greatly mitigated or kept in abeyance the paroxysms by this practice, particularly when the fits were associated to hysteria. By checking in this manner the attacks, I have been able to relieve seemingly hopeless cases.

Cauterization and setons to the nape of the neck go a great way towards hastening recovery from epilepsy. Blisters are strikingly effective when applied to the hypochondria in uterine epilepsy, or to the epigastrium when any dyspeptic trouble is connected with epilepsy. The following curious cases, which I will endeavor to state briefly, support the powerful influence of peripheral counter-irritation to avoid the epileptic fits, as pointed out by Brown-Séquard. A lady, belonging to a family in which most of the members had nervous disease, became epileptic at the age of puberty, the attacks gradually increasing until repeating almost every week. She had been under the care of my friend the late Dr. John Watson, who sent her to me in May, 1863. The treatment I instituted afforded very slight relief. Pressed by one of her relatives, this lady consented to the empirical and continued application of a large blister to the left arm, and thereupon the fits ceased.—An engineer, aged 28, subject to epileptic paroxysms followed by mania, lasting sometimes over a week, consulted me in April, 1866. Large doses of bromide of potassium and conium, actual cautery to the cervical region, shower baths, packing in the wet sheet, with regular nutritious diet, brought about great amendment in the

severity and frequency of the fits, no longer attended with insanity. Blisters at first and afterwards an issue, maintained in one of the arms, have kept the fits at bay for the last four years, allowing this man to resume his former occupation. In this instance the cauterization of the neck induced an eruption of large boils in the vicinity, which gave much inconvenience and pain to the patient, and hence my determination to apply counter-irritation to the arm.—In the third instance I notice, the arrest of the attacks has been accidental. A girl twelve years of age, for two years epileptic from unknown cause, came under my care in March, 1869. She was undergoing treatment, with slow improvement, when on the evening of the 10th of August she upset a small kettle of water, boiling over a gas-burner in her mother's room, and burnt herself severely on the right fore-arm, the chest, and also on both legs. From that day the fits, previously occurring two or three times a week, have not reappeared. We know that Andral and other eminent physicians advise the application of counter-irritation to the extremities in preference to the neck or head, in cases of epilepsy. Bona, Marshall, Claude Bernard, Delasiauve, and others cite cases of epilepsy cured by accidental burns. Conversely to theirs and the above cases, I may note this : A female at the New York Hospital for Epileptics was seized with a fit on going to lift a boiler with soup over the dining-room stove, and fell down, scalding her right arm, side, and neck with boiling soup. The paroxysms persisted

with their previous frequency, and were in no way influenced by the extensive burns accidentally produced in the trunk and limbs. I should not pass unnoticed case XXIII., in which blisters around the right leg would prevent the motor aura radically curing the fits. There is at the Hospital a girl, aged 17, who had never menstruated. She was seized with fits when twelve years old, after typhoid fever and diphtheria. Her body, small but well developed, displays the remnants of left hemiplegia in weakness, with appreciable diminution of volume of the left leg. The genitals are externally naturally developed, as also the vagina, which ends in a cul-de-sac with a central opening of the os externum, but there is no neck, and the body of the uterus appears reduced in length about one inch. The ovaries were very tender, and the mammæ, rather out of proportion to the size of the girl, exhibit sufficient glandular tissue but a very embryonic and irregular nipple. Persistent use of blisters to the ovarian regions have established the long-delayed menstrual flow. The fits had not recurred for over three months, when she was seized with a violent one prior to the onset of menstruation, and, I may add, that bromide of potassium has been administered in the case in doses of two scruples thrice daily.

Next to actual cautery,—so much dreaded by patients,—which operates deeply on the spinal cord, and with powerful effect on the fits, setons are most valuable to maintain counter-irritation to the neck. The operation can be performed without any pain by local

anæsthesia with Richardson's spray. Rhigolene is excellent for the purpose; and I place, instead of the common twist of threads for setons, a few silver wires twisted together. This metallic string keeps up the required irritation without needing renewal; the seton thus applied is easily dressed, does not induce the ugly inflammation or eruption of boils, often observed after the insertion of the twist of threads, while it allows the patient to bathe or shower the back, to dry the parts, and avoid offensive smell from decomposition of the discharge, which happens when the threads become moistened, even by simply dressing the seton. If irritation should subside, it can be quickly increased by drawing the silver string a little every day, or by smearing it over with some irritant ointment. I usually insert the seton, or apply the cautery at the level of the seventh cervical vertebra, to establish counter-irritation nearer to the sympathetic plexus, which accompanies the vertebral artery on entering the foramen in the transverse process of the sixth cervical vertebra, and to the inferior cervical ganglion. Cautery and setons are essential remedies in petit mal, or when mental derangement aggravates the effects of the fits; and, there is not an obstinate case, or any in which medicinal treatment had been tried fruitlessly, where I do not resort to counter-irritation, and the results justify me in this practice.

The evils of blood-letting in apoplexy are self-speaking, while the harm that it has done in aggravating those of our epileptics, who had recourse to it before

coming into the Hospital, has been no less striking. I never saw rational grounds to bleed in epilepsy, nor did I ever fail to counteract the cerebral congestion that may follow it, when dry-cupping of the back, active catharsis, and the derivation of the circulation from the head, were employed without hesitancy.

Whatever opinion theoretical writers might have advanced on the use of chloroform in epilepsy, I believe that few practical physicians after having tried it are willing to place reliance on it. I may assert that chloroform, instead of producing rest after its effects are over, leaves the epileptic not only exposed to vomiting and prostration, but as subject as ever to renewed convulsions and to excitement. In vertiginous epilepsy, inhalations of chloroform may prove fatal, as pointed out by Moreau. Fix, on inquiring into the value of inhalations of chloroform to detect epilepsy, saw them twice to aggravate the fits; Decaisne has met with three cases where they increased the severity of the attacks, and Tosquinet * is of opinion that they induce the paroxysms. As to myself, I am almost ever unwilling to resort to inhalations of chloroform in epilepsy to arrest the frequency of fits, or to afford quietness. Indeed, I do not recollect of any epileptic falling into many successive fits, or with maniacal excitement, who was relieved by inhalations of chloroform or ether. I am well aware that many epileptics guard off their attacks by inhaling chloro-

* De l'Inspiration du Chloroform comme Moyen de Constater l'Épilepsie. Archives Belges de Médecine Militaire. Tome xii.

form, yet, under such circumstances, chloroform not only operates by calling into action the reflex faculty, but as a stimulant of the spinal cord. Such patients, therefore, avail themselves of the excitation which precedes the stage of anæsthesia, without ever becoming insensible. In regard to chloral, I have found it a valuable hypnotic (twenty to thirty grains) in epileptic insanity; the calm it produces has often averted fits preceded by mental excitement, but—beyond this—I have observed no other preventive effects of chloral in epilepsy. I profit by the occasion to say a few words concerning one of the simplest ways to stop the fits, urged by Marshall Hall and Brown-Séquard, though not seldom untried. This means is, to dash cold water on the face of the patient at the inception of the attack, which at once produces, by reflex action, inspiratory movements with immediate change on the cerebral circulation. This plan undoubtedly shortens the fit, or suspends it in most instances.

I will not spend time in praising this or the other kind of bath, but will establish as an acknowledged fact, that any hydrotherapic course which will render more regular and active the peripheral circulation will prove beneficial in epilepsy. Cold shower-baths should not be prolonged beyond two or three seconds, and should be followed by active friction of the body, gentle exercise, or some stimulant, if reaction does not readily set in. The weaker the epileptic the shorter the shower bath should be, especially in the

beginning, and the popular practice of taking it for several minutes is highly injurious. I never saw any good from shower-baths lasting over a minute. Two or three seconds are the average length of such baths, which may be repeated twice or thrice daily. A tepid bath, of twenty minutes or half an hour, followed by a cold shower on the head and back, and active friction of the body, has operated very successfully in many cases. A similar plan has been advocated by Dr. Schreyer, of Hambourg.* He, however, keeps the patients for half an hour in a tepid bath at 28° Reaumur, and upon it pours two or three pails of cold water over the head and back; saline purgatives and digitalis, from four to twelve grains, being daily administered in conjunction with the baths. Alkaline baths, with carbonate of soda or potash, at 90° or 95° Fahr., cause a gentle reaction of the skin, with greater activity of the system, thus rousing the peripheral circulation, with an improvement chiefly marked in females, struggling feebly through the fits and disorders of the catamenia. These baths, taken at night before retiring, are very soothing, and afford quiet sleep in cases where the epileptics are troubled by wakefulness, nightmare, or dreadful dreams. Neither at the Hospital nor in private practice have I met with the success proclaimed by Chapman, from the constant application of ice to the spine to cure epilepsy. This application of ice I consider, therefore,

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* Revista Clinica di Bolonia. Junio 30, 1865.

very uncertain, and not more useful than any of the other hydrotherapic means.

I could not tell of what great advantage packing in a wet sheet may be in epilepsy to induce quietness and sleep. It is during the stage of excitement and restlessness subsequent to the paroxysms, or in epileptic insanity, that this means surpasses in its effects all narcotics. The pulse rapidly falls, becomes more regular and natural, general perspiration starts, and, if undisturbed, the patient thereupon goes to sleep in a short while. I have remarked many times the pulse over 100 brought down to 70 or 80 in less than half an hour after the wet pack, which thus operates as a sedative where other remedies are scarcely available. Its execution is not difficult. Even where epileptic maniacs do not exhibit submission to their immediate attendants, as is frequently the case, they can be nevertheless undressed easily and wrapped up in the wet sheet to be packed with blankets, in which they are bound without any power of action. I attended, with Dr. L. B. Edwards, a young gentleman subject to fits followed by most violent mania, and it was surprising to us how this patient would submit to be packed in the wet sheet, whereas he would oppose himself obstinately to any other kind of baths. I do not usually prolong the pack beyond two or three hours, and never disturb the patient when he or she goes to sleep. In many cases the packing has been renewed two or three times through the day with tranquillizing effect. The powerful sedative that we possess in the wet pack has

been recommended in strong terms by Dr. Robertson of the Sussex Asylum, and by Maudsley, who says: "Packing in the wet sheet has not only a soothing effect of itself, so that the patient will sometimes go to sleep in it, but, by keeping a restless and excited patient quiet, it enables sedatives to take effect when they would be perfectly useless if no such means were used." *

It is a commonly received opinion that epileptics ought to observe a low and severe diet, abstaining from animal food as well as from stimulants and coffee. And yet, necessity of nourishment has been evinced by those who have demonstrated the depressed state of epileptics, and chiefly by Brown-Séquard and C. B. Radcliffe. I feel convinced that I have benefited epileptics by insisting upon a systematic nutritious diet more than by the use of medicines. I have closely watched many epileptics in a hopeless condition, with their malady untrammelled by any drug, and have seen them very materially improved, thanks to a generous diet, whereas I know of no benefit reported from the starving treatment. And, here let me remark—how strange it is with those who advocate such plan, to overlook that the blood regulates nervous action, and that no blood is fit to nourish unless provided with proper elements, which are only supplied by good alimentation. The truth of this assertion was evident at the Hospital for Epileptics and Paralytics: most of the patients received, on open-

* The Physiology and Pathology of Mind, page 436.

ing the Hospital, were epileptics transferred from the Lunatic Asylum. Their condition was very low, and the first step I took was to establish a dietary table with meat every day, and other food in which nitrogen was abundant. Stimulants were allowed to the most enfeebled patients, and the improvement consequent on this course was manifest in the great diminution of fits and livelier appearance of the patients. I am fully aware of the frequency with which epileptic paroxysms supervene upon indigestion, or indulgence in stimulants, but I do not advocate immoderate eating or drinking. I only oppose abstinence and imperfect feeding in epilepsy. The craving for food of epileptics shows to the physician that it is a call of nature that must be attended to. It would be impossible to fix how much the epileptic should eat at a meal; neither should he eat as much as he wants. The plan so wisely advised by Brown-Séquard is undoubtedly the best: let epileptics be fed several times, and a little at each time, through the day. Hearty meals and rich food are always unwholesome. Meat should be preferred to farinaceous or other vegetable foods, on account of their difficult digestion and insufficient nutritious elements. The meat should contain fatty matters requisite to nourish the nervous system, and to keep the regular action of the bowels. By ordering the patient to take four meals—the last a couple of hours before retiring—we may devise a method suitable for most of the cases. Many epileptics experience nocturnal attacks, from the enfeebled condition in

which they run by leaving ten or twelve hours between the last meal of one day and the first of the next. Others, again, in the middle of the night are wakeful and very weak: by taking some nourishment before going to bed this trouble is easily prevented. As long as the hungry feeling subsists, we may foretell the recurrence of fits, for one of the first signs of permanent improvement is a natural appetite, without any longer experiencing the unsatisfied desire to eat, exhibited by epileptics. I, as a rule, forbid to epileptics alcoholics between meals, or free allowance of wine with these latter. Coffee answers as an excellent stimulant without the objections attached to alcoholics. I have carefully inquired into the effects of coffee in epilepsy, and I have every reason to believe that it does not produce any of the evils generally deprecated. On the contrary, epileptics accustomed to it experience a comfortable feeling, and ask for it to rouse themselves from weakness or depression. If administered immoderately from the beginning, it may not be borne with ease; however, coffee should be mainly ordered with the meals, to avoid thereby unpleasant effects. I have previously referred to the usefulness of coffee to counteract the drowsiness and to retard the effects of bromism, induced by large doses of bromide of potassium.

I must now allude to the importance of bodily exercise, it being extremely advantageous, besides a proper diet, that epileptics will combine amusement with suitable gymnastics. The practice of calisthenics,

which I introduced at the Hospital, though carried out imperfectly, has rendered, nevertheless, a great deal of good. Epileptics should not be permitted to remain in idleness; they ought to be made to move about, if they can but walk. Gymnastics, tried as far as the strength of the individual will allow, are of much avail, for, by systematically training him or her, we may deeply change the constitution and subdue the nervous system. The exercises should never be continued until fatiguing the patient, lest they might prove injurious. One of the great dangers in the *movement cure* is the frequency with which the patient's endurance is exerted beyond limits of healthy reaction. In private practice the obstacles to make epileptics exercise are only realized by those who have to contend with their lack of energy and of determination to do anything. In hospitals the difficulties are no less, for epileptics accustomed to a life of indolence are always reluctant to engage themselves in any occupation. Yet, it is needful to their welfare that they devote themselves to some kind of labor, and that they be equally provided for in their intellectual advancement. I have witnessed unquestionable benefit from the physical and moral training of epileptics, which I have endeavored to have established at our Hospital.

To close with what concerns treatment, I will now relate three cases where I have performed the operation of trephining the skull for the relief of epilepsy.

Case XXXII.—*Epilepsy occurring fourteen years after injury to the skull, with incomplete paralysis of the left facial and sixth nerves, and diminished sensibility and temperature of the skin, extending to the arm on the same side; tonic contraction of the right muscles in the cervical region, with lateral distortion of the head; great frequency of the pulse, and polyuria. Removal of part of the occipital bone with inner exostosis pressing upon the brain, violent epileptic convulsions when the trephine penetrated the diploic tissue.*

The following notes are taken from records carefully kept by Dr. L. B. Edwards.

The patient, male, 21 years of age, born in Johnston, Rhode Island, unmarried, and by occupation a clerk, placed himself under my care the 15th of January, 1868. Height five feet ten inches, weight 145 pounds. Complexion dark, eyes and hair black, beard thick. Head regularly shaped, high forehead, limbs well proportioned, muscular system strongly developed. Parents both living now, in good health, and none of their family have had epilepsy or insanity. Has since childhood a right oscheocele. Infantile and adult health perfectly good, with the exception of an attack of typhoid fever in the autumn of 1863. Habits regular. No venereal excess, or syphilis.

In August, 1852, being six and a half years old, he fell from a low swing and deeply wounded the scalp, just on the left of the occipital protuberance. Remained unconscious for a while, but apparently recovered completely. The cut bled very freely, did not heal up in six weeks, on account of exuberant fungosities, and left a transversal cicatrix, extending about two inches to the left of the occipital protuberance, rather abundant with inodular tissue of a dull rose color, painless, and of a fibrous consistence and hardness. Suffered from severe and persistent headache after the fall, and continued thereafter subject to it, the ache being general over the head. In 1862, was troubled with seizures of sudden jerkings and spasms of the arms, they being stretched out and involuntarily thrown above the head, making objects fall out of the hand. These seizures were particularly observed in the morning, after arising from bed; is unable to recollect if they troubled him on any date prior to the above. Has never had any giddiness or vertigo, nor does he remember having ever awaked with headache or sore tongue, or wetting his bed at night. The first epileptic attack happened early in the morning in July, 1865, without any circumstance which can be regarded as its exciting cause.

It was ushered in by the acute cry which has invariably preceded all subsequent attacks. These were repeated five times during the three years following the initial one, but now come on every third or fourth day. The jerking and twitching of the arms never ceased during the period intervening between the attacks, and always with their morning character. Originally the convulsive paroxysms took place also in the morning, whereas at present they fall upon him at any hour of the day or night; the earliest four or five immediately followed by vomiting, not now present, and on no occasion has he had the least premonition of the coming convulsions.

When I first examined him, the 15th of January, he had just got over two severe attacks, which occurred in the course of the previous twenty-four hours, and his condition was as follows: countenance rather pale, pupils of equal size and enlarged, slight though perceptible converging strabismus of the left eye, minute petechiæ on forehead and eyelids. Incomplete left facial paralysis, with distortion and elevation of the right angle of the mouth. Muscles of the paralyzed side flaccid—if an attempt is made to blow with mouth shut the left cheek soon yields to the effort; the saliva does not dribble, however, from the mouth, and he can whistle without difficulty. But the tongue—not furred—when protruded deviates to the left. Soft palate, firm and natural. Speech never embarrassed Hearing, smell, and taste unimpaired. Excepting the occasional double vision from the strabismus, there is no other derangement about his eyes. The æsthesiometer shows sensibility deadened on the left side of the face and throat, no loss of feeling in either half of the tongue, with papillæ small and red. The temperature (90° Fahrenheit) is two degrees lower on the left than on the right side of the face and neck. Head inclined to the right; on this side the trapezius, splenius capitis, and complexus, are in rigid contraction determining the lateral distortion, with prominency of the integuments on this part of the cervical region. No evidence of paralysis, numbness, or abnormal sensations in the upper or lower limbs, nor is there any difference in the comparative size of their respective muscles; but when the epileptic paroxysm is over, the arms remain powerless for a shorter or longer time. Sensibility and temperature are less on the left than on the right arm, which is two degrees warmer (88°Fahrenheit). This difference is more evident at the hands, usually cold, and inclined to turn livid. No such diminution of sensibility and temperature is detected on the legs, excepting, however, the feet, which are cold. Walks steady even with eyes closed. The temperature over the cicatrix of the scalp is three de-

grees more (92°) than that of the rest of the head. The increased heat is quite perceptible to the hand laid over the spot. The cicatrix, as already remarked, is not painful; in fact, it has never before attracted his attention. Three-quarters of an inch above the superior curved line, and more than one inch to the left of a line prolonging the occipital crest, directly under the cicatrix and firmly united to it, the cranium presents an irregularly delineated elevation, rough to the touch and painless upon pressure. If this spot is sharply percussed with the fingers, the patient feels dizzy, seeing before him as if it were "a large white plain with a central black spot"—this percussion, even though tried several times, does not awaken such a sensation on any other part of the cranium, but always starts it when repeated over the cicatrix. Pulse quick, firm without tenseness, not uniform, and ranging from 108 to 120, according to different observations at distinct hours of the day. This frequency greatly abates after the paroxysms; I found it 87 after the attack shortly to be described. Heart sound, increased in activity; there is no oppression, nor feeling of faintness; chest well formed, lungs equally sound, respirations 20 per minute. Digestive functions regular, seldom inclined to costiveness. Appetite good. Drinks considerable quantities of water. Remarks that he passes a large amount of urine, and that he has done so long before this. Has been on a few occasions troubled with erections after the attacks. Careful measurement and investigation, carried on by Doctor Edwards, show the quantity of urine voided in twenty-four hours to be 210 fluid ounces. The liquid at a temperature 82° Fahr. was perfectly transparent, of a pale amber color, without any flocculi. Specific gravity 1,010. Acid reaction. No evidence of sugar with Trommer's test. Abundance of chlorides.

The foregoing description needs to be completed with an account of symptoms during the paroxysm. I will presently narrate them as they were during one attack I witnessed. He was at the moment playing whist, and I happened to engage him in conversation as he was shuffling the cards. Suddenly he stops his discourse; the cards drop out of his hand; becomes pale, unconscious, eyes fixed and converging, with pupils largely dilated, and then gives out a prolonged expiratory cry, concomitant with increased lateral distortion of the head, and rotatory movement of the body to the right, with arms firmly flexed on the chest, and mouth wide opened by spasmodic contraction of the muscles of the neck. Cold water dashed on the face roused him at once out of this condition; but the arms remained twitching, his countenance lost all expression, the eyes

rolled continually, did not hear what was said, smacked his lips, and there was a strangeness in his manner as consciousness gradually returned, with relapses into momentary oblivion, to be entirely recovered in about twenty minutes. Along with this last stage there was profuse perspiration of the face and hands and repeated contractions of the diaphragm, with efforts at deglutition and raising of frothy mucus. There was, in addition, an increase of facial paralysis, strabismus, and lateral distortion of the head, and the arms hung powerless for some time after the paroxysm passed over, without leaving any headache or drowsiness. The pulse was 87, extremely irregular, and at moments imperceptible. As on former occasions, there was not the slightest premonition of the attack, neither knowledge nor remembrance of it, and every symptom corresponded with those related by his father in connection with the other fits. Furthermore, Dr. Edwards had a new opportunity of observing again identical phenomena during another attack, which occurred the next morning, while the patient was about being packed in the wet sheet. In reference to the attacks, I must add that their frequency has made memory defective, and the patient's disposition very irritable and overbearing. It is unnecessary to state that he has resorted to every kind of remedy for epilepsy. From August, 1866, to last January, he discontinued eating meats; also went on a long pedestrian journey, during which all rules as to diet were disregarded, and for eleven months before July, 1867, he had no fits, although the morning twitching of the arms persisted. Meantime he used bromide of potassium, and has faithfully continued with it to the present day, now exhibiting on the face and body the peculiar eruption induced by the salt. Since the above date, the attacks, with increasing tendency and detriment to the mind, have incapacitated him from any pursuit.

From the preceding symptoms, I judged without hesitancy that the injury sustained by the skull was the unsuspected origin of the epilepsy in question. I supposed that a growth on the inner table of the occipital, corresponding to the external cicatrix, was the very source of all derangement; consequently, persuaded that no medical treatment could ever be capable of eradicating the structural changes undergone by the bone, I thought the best course would be to remove the evidently diseased portion of the occipital, and expressed candidly this view to the patient and his father, with the serious risks of the operation, which I would not, however, undertake unless acknowledged justifiable on con-

sultation with some other physician. Waiting for further decision, the case was kept on observation, and ordered the following mixture:—

Potass. bromidi gr. xxx., ammon. brom. gr. vj., liq. potass. arseniti ♏ iij., Aquæ dest. f. 3 ss. Misce. To be repeated three times a day; in addition, to take, morning and evening, one pill with: Ergotinæ, ext. gentianæ, āā gr. ij., pulv. acaciæ q. s. Ut fiat pilula.

He was to be packed every morning in the wet sheet for three-quarters of an hour, before going under a cold shower bath for six or eight seconds, and which should be repeated in the evening.

On February 2d, my friend Dr. L. A. Sayre was called in consultation, to which Dr. Edwards also assisted. Having heard the history of the case, and carefully investigated the condition of the patient, Dr. Sayre observed the very same symptoms before detailed. He distinctly detected the irregular elevation on the left side of the occipital, under the cicatrix of the scalp, and corresponding to a smooth surface on the opposite side. The heat of the part was most manifest to the hand, the thermometer marking at this examination 94°, whereas it did not go beyond 90° in other regions of the head. The diminished sensibility and temperature on the left side of the face and hand, the facial paralysis, strabismus, and lateral distortion of the head, with contraction of the muscles on the right side, were again noticed. The patient was equally explicit as to the sensation experienced when the cicatrix was tapped with the ends of the fingers. His pulse was 110. Respirations 18 per minute.

I was gratified to find that my diagnosis agreed in every respect with that of Dr. Sayre; consequently, he asserted that, in his belief, no medical treatment could be available unless the degenerated portion of bone —which was the true source of trouble—were removed from the skull, and that this operation, though serious, was, however, the only way capable of reaching and most probably curing the disease. Endorsed by such an unreserved opinion, and with previous consent of the patient, I determined to operate. Therefore, assisted by Dr. Edwards, I proceeded to remove the portion of bone affected, on February 10th, at 3 o'clock P.M., weather being very clear and cold. The patient took a very light breakfast in the morning, had bowels thoroughly relieved by an injection, and the hair from the back part of the head cleanly shaved, previous to the operation. Ether was administered, and anæsthesia shortly completed after the usual period of excitement. He was laid on the right side, with head resting on a hard pillow. A perpendicular cut, about two inches, was carried down to the bone, and crossed at right angles by

another horizontal incision of nearly three inches, running from the external occipital protuberance to the left. The incisions thus made bled quite freely, the flaps were dissected, a small branch of the occipital artery twisted, but hemorrhage from other vessels continuing required us to mix alum with the ice water in which the sponges were wrung out, and to use compression to arrest the loss of blood. On exposing the periosteum, it was found very much thickened, highly vascular, firmly adhering to the cranium and interspersed with hard granulations of a dark crimson color. The bone was scraped, and Galt's trephine applied, fixing the perforator in the middle of the bony eminence. No sooner had the instrument bored through the hard outer table and penetrated into the diploë, than the patient was suddenly thrown into an epileptic fit. He did not utter the peculiar cry of other attacks, but the limbs were rigidly stretched out, his body rolled over to the right, bit his tongue, had a great deal of froth at the mouth and venous congestion of the face, with deep snoring and, lastly, relaxation of the limbs. During this time, the operation had, of course, to be suspended: the bone was then bleeding most profusely. The stertor and other signs of the fit over, the trephine was reapplied: most of the cone penetrated through without loosening the bone; on attempting to raise the disc with the elevator it gave way, leaving behind a resistant portion at the bottom of the perforation. Hemorrhage increased at this moment, and, indeed, blood gushed out with violence from the vessels of the diploë. Cold alum water had to be steadily applied, and the opening plugged before application of the trephine to the portion left could be renewed. The elevator was then again and again tried after gentle turnings of the trephine; but the irregular shape of the protrusion made the bone, less and less resistant as we approached the dura-mater, break at every effort, and it was in this wise removed in small fragments. The last of these fragments were united by adhesions to the dura-mater. At the internal part of the opening made by the trephine there was a conical indentation, pressing on the brain, and close to the superior longitudinal sinus. To take away such indentation, with its broad base and the abnormal looking bone around the perforation, was the most tedious stage of the operation. It had to be achieved with the bone-nippers and the lenticular knife, in a very slow, careful way, holding up at every cut to stop the incessant hemorrhage. Finally, all the apparently unhealthy structure was pared off from the occipital bone in an oval space, two by two and a half inches in diameter, and the finger gently slipped around between the membranes

and the bone showed no irregularity of the inner table pressing on the brain. The membranes looked healthy and uninjured, excepting at the very small site of the erosion, caused by tearing the adhesions to the exostosis. The longitudinal sinus, uncovered about an inch, could be seen projecting inside the cranial opening, and along the lower edge of this opening the lateral sinus could also be felt. Every portion of thickened periosteum was excised, and when the raw surfaces ceased bleeding, the flaps were brought together by deeply inserted silver sutures, exclusive of the lower incision, that was closed with serrefines. The operation was completed in three hours and a half, and no more than six fluid ounces of ether were inhaled by the patient. The anæsthetic effects were soon dissipated, bringing about vomiting, which, with intermissions of from twenty to thirty minutes, persisted until two o'clock in the morning. Ice was constantly applied to the wound, and the following mixture exhibited:—Sp. ammon. arom. ♏ xx., aquæ f. ℥ ss. Misce.

February 11th. Slept tolerably well, after vomiting discontinued. Pulse this morning, 132-136, irregular and full. Skin dry and hot. Tongue slightly coated in the centre. Complains of being very thirsty, and finds great satisfaction in swallowing small pieces of ice. Urine in nearly equal quantity and with same reaction as before the operation Ice-bag kept all the time to the head. Wound dry, with lips of a slightly red color. Diet: beef-tea, every two hours.

February 12th. Was restless last night, frequently wishing to be turned in bed, and talking in his sleep. Pulse 140, full, but not firm. Tongue furred, with red edges. Skin hot and moist; has perspired during the last hours, and at 11 o'clock A.M. had a long chill. No pain in the head; wound sensitive, but without redness, swelling, or heat. Ordered the same diet, and to take in the evening: Potass. bromidi ʒ ss., sp. ammon. arom. ♏ x., aquæ dest. f. ℥ ss. Misce.

February 13th. 4 o'clock A.M. Pulse 143, after disturbing him in bed. At 8 o'clock A.M. it was 115, irregular and soft, and came down to 105 during the evening. Ice-bag continued to the head. No discharge as yet from the wound; the edges seem united by first intention, excepting where the serrefine was inserted. Diet: beef-tea, soup, gruel.

February 14th. Has slept well through the night; at 9 o'clock A.M., pulse 105, and weak. Sutures removed—wound gives no pain; ice has been incessantly applied to it, and the patient is in excellent spirits. Bowels moved with turpentine and assafœtida injection. Tongue clean and natural; appetite good. Diet: eggs, soup, rice.

February 15th. Pulse 102, soft. Bowels opened through the day. The incisions are healed up, excepting at the very point of their crossing, through which oozes a sero-purulent discharge. Continue ice to the head.

February 16th. Same treatment. Pulse 98, soft. Bowels moved naturally.

February 17th. Pulse 92, weak, not uniform. Wound still discharges a limpid serosity from the centre. Treatment and diet as above; bowels operated once this day.

February 18th. Pulse 80, regular and soft. Can now lie on back of head without discomfort. Central crossing of incisions still open. Wound dressed with glycerine and Venetian turpentine. Local application of ice discontinued; same diet.

February 19th. Pulse 71, soft and small. Wound dressed as yesterday; slight discharge. Bowels moved to-day. Urine reduced to three pints in twenty-four hours. Set up for an hour in the afternoon. Diet: soup, roast beef, and coffee.

February 20th. No change to be noticed. Pulse 73, firmer and regular.

February 21st. Pulse 75, regular and larger than before. Slept very well all night. Opening of the wound completely closed. Same diet. In the afternoon gets irritable and fretful, from not having his own way.

February 22d. Was restless last night. Wound feels sore. Pulse 92, contracted and irregular. Felt better in the afternoon; sat up, and being alone with his assistant, took a whim to go out of his room. Complained that such undue over-exertion "tired him out." Had a chill, headache, and convulsions at 3 o'clock P.M., whilst sleeping in an easy-chair. The wound, which had been swelling, bled considerably, and became excessively painful and hot. Had great pains across the head. Pupils dilated, cheeks congested, tongue furred in the middle. Pulse, after convulsions, 81, irregular and soft. Ordered *illico:* turpentine and assafœtida injection, ice to the head, and mixture of potass. bromidi gr. xl., sp. ammon. arom. ℥ x., aquæ dest. f. ℥ ss. Misce, to be repeated in the evening.

February 23d. The above symptoms persisted until this morning. Has been very fidgety; the convulsions returned twice—at 10 o'clock last night, and this morning at 11 o'clock, but less severe than the first. The wound being quite distended, I opened it at the middle, with a probe; a dark, bloody, purulent discharge came out, and from this moment the general irritability, headache, and other symptoms abated. Ordered: ergotine pill, and mixture brom. potass., as before the operation; turpentine

and assafœtida injection; ice continued to the head; light diet and coffee.

February 24th. Pulse 78, regular and small. Says "he feels all well." Same treatment. Diet: soup, beef, and coffee. Wound discharged this morning about an ounce of pus, and has run all day.

February 25th, 26th, 27th. The suppuration is lessening every day. Pulse from 76 to 78, regular and firmer. Same treatment and nutritious diet. Head bathed every morning with cold water, and local application of ice maintained to the wound. Urine has not exceeded 72 fluid ounces in 24 hours. It is transparent, acid, without sugar, still abundant in chlorides, and with specific gravity 1020.

February 28th to March 6th. The improvement progresses. The discharge decreases, and it only amounts to a few drops of lymph and pus. Incisions entirely cicatrized, with the exception of the small fistulous opening at their crossing. Pulse regular, 76; bowels act every day. Continue with ergotine and brom. pot. Nutritious diet. Says that he feels more the impression of cold water on the right than on the left of the top of the head. This morning, whilst pressing out the discharge from the wound, had a strange feeling in his head, as though the blood rushed to it, without, however, losing consciousness, and burst into a fit of crying, with deep sighing. This condition passed away in a short while, and was certainly the result of the cerebral pressure. The signs of facial palsy, strabismus, and distortion of the neck have disappeared. The quantity of urine remains unchanged.

May 7th. On March 18th an abscess appeared under the old cicatrix of the scalp, with drowsiness, vomiting, and convulsive symptoms. A free incision let out a large amount of pus, the symptoms subsided, and the sinus left was dressed with Peruvian balsam. The parts soon healed up, and counter-irritation was applied to the back of the neck. However, the uncontrollable disposition of the patient, with the excitement due to a visit of his friends, brought about the recurrence of headache and convulsions on the evening of April 13th. No further derangement has occurred since that date; the cicatrix of the scalp is quite firm and painless, and the paralytic symptoms and polyuria have no longer existed. He continued the same course of treatment, the bromide of potassium being raised to a dose of forty grains, to be taken three times daily, before bringing it down to a half of that quantity.

I have not heard of any recurrence of the epileptic attacks since the day above mentioned.

CASE XXXIII. *Epilepsy from fracture of the skull. Trephining at the site of injury, and removal of an old standing clot, with immediate recovery of the intellectual faculties.*

(From notes by Dr. D. McEwan, Assistant Physician to the Hospital.)

J—— C——, aged 22, was admitted into the Hospital for Epileptics October 1st, 1867. Ten years ago he was engaged in gathering wood from the second story of an unfinished building, and while trying to escape from a workman who was pursuing him, fell through a window headlong upon the flags of the pavement below. The fracture of the skull which resulted extended upwards across the right parietal bone, the fragments displaced leaving at the lower part a space deficient in bone, forming a depression where marked pulsation could be detected. Six months after infliction of this injury, the epileptic attacks commenced, with the nocturnal character they have preserved throughout their frequent recurrence, preceded by a cry, and impairing the intellect until rendering the patient almost an idiot. Since admission into the Hospital he had been unsuccessfully treated with every variety of remedies, having from ten to twelve fits every month. This circumstance, and the nature of the injury received on the head, the displacement of bone, with the dull stupid condition of the patient, who scarcely ranked above a helpless idiot, decided me to trephine the skull, in order to relieve the brain from the irritation of a clot which I supposed to exist at the site of injury, as I manifested to my class when the patient was brought to my clinic at the University, Sept. 28, 1869. The patient was therefore etherized, and the operation carried out Oct. 2, at 1 o'clock P M., in the presence of Professor Boëck, of Christiania, Dr. M. Clymer, and Drs. McEwan and Morgan, Assistant Physicians to the hospital, and other visitors. A portion of the parietal, three inches long by nearly one inch wide, removed from the upper extremity of the fracture downward to the above noticed depression, allowed me to withdraw from a cavity underneath an old sanguineous clot, in a state of fatty degeneration, embedded in the brain, and measuring one and a half inch long by nearly one inch in diameter. The walls of the cyst were white, shining, slightly vascular, and formed by the very cerebral tissue which had suffered such a deep laceration. The bleeding was abundant in opening said cavity, from division of a branch of the middle meningeal artery. Hemorrhage was stopped by the actual cautery. The edges of the wound were brought together by a few silver sutures; constant application of ice was ordered to the wound, and a pill

of ext. conii and ergotine, āā gr. ij., administered regularly every hour, while the patient was awake. On recovering from the effects of ether, at 5 P.M., he appeared very much excited, and began talking loudly and fluently. The scene around him, just previous to the operation, seemed vividly impressed on his mind. He described with great accuracy the sensation produced by the ether, his forcible detention on the operating table, and the appearance and dress of some of the physicians who held him. He seemed particularly anxious to return from the separate room where he was, to the pavilion among his companions. An ice-bag being applied to his head, he complained of feeling cold all over. A few teaspoonfuls of whiskey were given, and he was wrapped up warmly in bed. Pulse 72, somewhat bounding, skin cool, pupils partially dilated.

7 o'clock P.M. Took his first pill with some difficulty; very suspicious of narcotism and of undergoing "more operations." Chilliness persists. Ordered f. ℥ j of whiskey in water, which in the course of half an hour resulted in an improved state of the pulse, skin, and temperature.

10 o'clock P.M. Has taken three pills; pupils dilated, pulse 80, full and regular, skin natural. He is unable to urinate, although he thinks he could do so if permitted to stand up in bed. This not being allowed, the urine was drawn off with the catheter, after great objection and dread on the part of the patient. Slept none all night, talking in the same alarmed, excited strain, and exacting from his attendant that no further "operating" would be attempted.

October 3. 6 o'clock A.M. He is still very much excited, especially when he sees the Doctor, whom he regards with the very strongest suspicions. He was extremely restless and noisy during the night, objecting to the application of ice-bags to the scalp wound.

8 o'clock A.M. Pulse 78, full and regular; skin warm and perspiring; pupils still dilated; respiration normal. Passed the catheter again, drawing off about a pint of urine. He has had no nourishment during the last twenty-four hours. To have iced lemonade, beef-tea, and milk, but no solid food. Continue pills on every hour while awake, and ice to the head.

5 o'clock P.M. Pulse 92. Pupils dilated. Is still extremely restless and anxious. Took a very little beef tea and milk, but drank freely of the lemonade. Passed urine naturally twice during the day. Bowels moved after injection of Ol. Terebinth. f. ℥ j, Tinct. Assafœtida f. ʒ iv, ad Mucilag. Oj.

8 o'clock P.M. He has talked more within the last twenty-four hours probably than during the twelve previous months, and though he repeats himself a good deal, and is wild and extravagant in his ideas, yet when questioned on any subject within the range of his knowledge he will answer promptly and to the point.

October 4th. 7 o'clock A.M. Slept soundly all night, and is much quieter and more rational this morning in consequence. Pulse 80, full and strong; skin natural, pupils slightly contracted. The ice-bag was kept to his head uninterruptedly during the night. He is very anxious to ascertain this morning the object of these cold applications, and while it is explained to him listens with attention, occasionally interposing a few very pertinent remarks. His eye has lost its dulness, and his face the vacant stare formerly habitual to it; his whole countenance expressing now intelligence and mental activity. Nothing transpires in the room which he does not notice and remark upon; he speaks to every one who enters, and his queries and retorts at such times are lively and pointed, evincing that he has recovered his power of observation and reflection. He says that he will submit to the ice-bags, the pills, and any kind of diet, if Dr. McEwan will only promise that no further cutting will be attempted, and no more ether administered. When I visited him that day he received me with great mistrust and fear. He jumped up in bed, beseeching me not to come near him, and not until I assured him of my contrary intentions would he become tranquil. In consequence of this excitement, his pulse went up to 140, and continued to beat at the rate of 100 per minute even one hour afterwards.

6 o'clock P.M. Pulse down to 75. Skin, temperature, and pupils normal. Continue hemlock pills steadily, and give at 8 o'clock P.M. Potassii Bromidi, 3 grs., Fl. Ext. Conii, ℥xx, Syr. Aurant. Cort. ad f. ℥j. He has taken a considerable quantity of beef-tea and milk during the day.

October 5th. About 1 o'clock A.M. had a fit of the same character as his former ones. During it, and with each respiration, a little bloody serum would well out from the wound. Dressed the wound with lint, and ice-bags again applied.

8 o'clock A.M. Pulse 80, pupils natural. Talks more rational, is not so restless, but is still distrustful and anxious regarding his prolonged stay in a room by himself. Bowels moved freely after the usual injection of turpentine and assafœtida. Allowed him some dry toast and tea as well as a pint of milk this morning. At his earnest request one of his companions visited him during the day, and after a protracted debate, suc-

ceeded in tranquillizing him on the "operating," the removal of bone, and other knotty subjects. Consequently, he was prepared to receive my usual visit with calmness and assurance, if not cheerfulness, his pulse only taking a gentle rise to 102. Questions he answers with readiness and precision, and in so doing his command of language astonishes every one who knew him before; he also remembers clearly every event of any importance which has happened to him since October 2d, the date of the operation, and he often refers to conversations which he has held with different individuals since that time.

6 o'clock, P.M. Pulse 74, pupils again dilated; the pills of conium and ergotine have been steadily administered, one every hour while awake. To take in addition the sleeping draught with bromide of potassium, as above prescribed.

October 6th. 8 o'clock A.M. Pulse 72, pupils partially dilated; slept well during the night. The silver sutures were removed, and the scalp-wound seems disposed to heal by first intention, although a little puffy at the lower part. The application of ice to the head no longer causes pain, but only a feeling of numbness. The patient is cheerful and talkative this morning, without being noisy as on former occasions. The excitement, amounting almost to delirium, which followed removal of the clot irritating the brain has subsided. He is no longer noisy, suspicious, or impertinently curious, but he is as eagerly observative of persons and events, and as desirous of information on all possible subjects as a child who is just learning to speak. He was then questioned regarding the circumstances which led to his receiving the injury to the head, and gave information of the facts detailed in the beginning of this report. His remembrances of these events came slowly, and only after deep and earnest reflection. It baffled him for a long time to explain in particular one circumstance connected with the accident. He could not remember how or why he came to fall from a window while being pursued, and his inability to elucidate this point fretted him very much. An hour afterwards he called out, "Dr. McEwan," his face beaming with satisfaction, "I know how it was now, Doctor," said he—and then he went on to explain how, when the workman was chasing him, he ran "to chuck" his bundle of wood out of the window, so as to secure it from the pursuer, and how, in "chucking" it out hurriedly, he lost his balance, and knew nothing more. While narrating this, his face was wet with perspiration, and he unconsciously worked himself out of bed; so absorbed had he been in trying to decipher out these facts from the blurred page of his me mo

October 7th. The patient laughs at his former fears regarding the doctors. More nourishing food, such as eggs, beef-steak, and soup, was now allowed him—the treatment by cold application to the head, conium, ergotine, and the bromide of potassium being uninterruptedly persevered in. From this time forward he continued to amend; the scalp-wound healed rapidly without suppuration; his mental condition remained bright and promising, and on the thirteenth day after the operation he was found sitting in a chair, clothed in his right mind, and reading a newspaper, a feat which no one until then had supposed him to be capable of. On the following day he was allowed to walk about the grounds of the Hospital for an hour or two.

The foregoing injury sustained by the brain made me suspect that the operation would not prove sufficient to remove the fits, and I expressed this opinion to Professor Boëck and Dr. Clymer. My suspicions were soon corroborated, for the fits recurred again at night, at intervals of from ten to seven days, and have gradually decreased in severity and frequency. They have not induced any mental deterioration. Last February, after remaining for several days free from fits, he became morose, and in a sort of melancholic condition, from which he was roused with difficulty. He continued five or six days in this state, and completely recovered after having one of his regular nocturnal fits. Did epilepsy assume then its cerebral form?

It is, of course, uncertain what the final issue of the case will be, and I am very doubtful of ever arriving at a complete cure of the fits, although the harmlessness they have exhibited and their diminishing frequency are rather encouraging circumstances. The immediate recovery of the intellectual faculties, however, has been a remarkable and persistent improvement obtained by

the operation. Finally, I will state that the microscope displayed the fatty change undergone by the fibrine in the clot, and the decomposition of the coloring matter of the blood, which were otherwise manifest to the naked eyes.

Case XXXIV. *Epilepsy from injury to the head, attended with violent mania.—Trephining of the skull.—Cessation of the attacks.*

J—— Ch ——, aged 38, by occupation a carriage-driver. Grandfather insane, mother phthisical. Entered the Hospital in the beginning of October, 1869, and the following details are abstracted from notes taken by Dr. J. H. Morgan, Assistant Physician. The epileptic fits and mania directly followed severe contusion of the head, which occurred six years ago, the patient having been thrown from a carriage and struck with his head a lamp-post in the Central Park. No cicatrix existed at the site of injury, but the scalp and bone underneath were very sensitive and painful to pressure. The attacks were lately reduced to paroxysms of violent mania, persisting for several days, in which condition the patient was when he came to the Hospital. The operation of trephining the skull was performed, attended by Drs. McEwan and Morgan, and in the presence of Drs. R. L. Parsons, T. H. Kellogg, and other physicians. Ether was employed to produce anæsthesia, and a circular portion, near the postero-superior angle of the left parietal bone, over an inch in diameter, was removed. The bone was thick and hardened from inflammation. The patient recovered from the anæsthetic in such a wild condition, that it became necessary to keep him in the strait-jacket, and to use a subcutaneous injection of ten drops of Magendie's solution. An ice-bag was constantly maintained to the scalp-wound, and ergotine and conium administered, as in the preceding case. The subsequent progress of this one, extending until the end of December, took place with occasional paroxysms of mania, which rendered it necessary to resort to large doses of bromide of potassium, and of conium, packing in the wet sheet, etc. The wound suppurated very freely, upon being irritated by the restlessness of the patient, and his knocking the head frequently against the sides of the bed. The back of the head and neck displayed an eruption of boils. The maniacal excitement continued amending, and disappeared altogether upon exhibition of ten grains of chloral, three times daily. The

patient had completely regained his intellectual faculties, and asked his discharge from the Hospital in the commencement of January, 1870. He has had no recurrence of fits up to April 6, 1870, and has resumed his former occupation of driver. I fear, however, that irregular habits or drinking, in which the patient formerly indulged, might induce a relapse of mania, which is further incubated by hereditary predisposition.

I need not assert that, judging from my own experience in the matter, and from the analysis of cases reported by different authors, I am in favor of trephining the skull for the relief of epilepsy due to local injury to the head. I consider with Dr. Wm. H. Van Buren,* that the operation is comparatively a simple and harmless one, its seriousness being explained by the fact that it is usually performed in cases which from their nature are fatal. The risks of trephining the skull, under such circumstances, are greatly diminished if special care be taken not to injure unnecessarily the dura-mater and cerebral membranes. The testimony of the statistics by Stephen Smith,† embracing 27 cases; of Billings,‡ who gives a table of 72 cases, and of James Russell,§ who has grouped fifty cases in which the skull was trephined to relieve secondary nervous disease, afford a strong argument in favor of the operation, independently of any other consideration that might justify it. I find that, as in Case XXXIII., an old blood-cyst, of three years' duration, had deeply depressed the sur-

* Philad. Med. and Surg. Reporter, Dec. 29, 1860.

† New York Journal of Medicine, March, 1852.

‡ Am. Journal of Medical Sciences, July, 1861.

§ British Medical Journal, Nos. 232 and 233, June 10th and 17th, 1863.

face of the brain and seemed to be the cause of fits in one of the cases referred to by Russell in his valuable paper. I do not feel quite certain that simple depression, without any other injury of the scalp or cranial bones, or meningeal effusion of blood, be but exceptionally attended with epilepsy; the amount of pressure that the brain may thus sustain without any mental impairment or paralytic affection being, indeed, remarkable. The great sensitiveness of the bone in Case XXXIV. betrayed its diseased state, which was in no other way evident. It seems legitimate to suppose in Case XXXVII. that irritation with the trephine in perforating the exostosis was the true cause of the convulsions, most assuredly helped by the condition of the nervous centres in anæsthesia. The disc of the occipital, removed with the trephine, had an uncommonly dense external lamina—about four lines thick—covering a cancellated structure containing the canal of a large blood-vessel, about two lines in diameter. Putting the fragments of bone together, the exostosis was nearly one inch from the outer to the inner lamina, a thickness certainly very considerable, when we remember that at this site the tables of the occipital run closer and closer to each other, until the middle of the fossa for cerebrum, where there is scarcely any diploic tissue. The most solid parts of the bone removed exhibited neither Haversian canals nor lacunæ, and these latter, of irregular form, did not surround concentrically the canals in the less dense structure close to the diploë.

Now, as to the nature of the paralysis in this case. Was the incomplete paralysis of the facial and sixth nerves *reflex*, as Brown-Séquard admits to be frequently the case with cerebral affections? It is unquestionable that the peripheral extremities of the trigeminal, distributed to the dura mater and bone, were implicated in the modification of structure discovered at the injured spot. The frequency with which irritations of this nerve, like those of the sympathetic, are apt to originate incomplete, and the so-called direct paralysis, has also been pointed out by the above distinguished physiologist. When we consider the relations between the origin of the trigeminal, the facial, and the sixth nerves, we can easily understand how excitations conveyed through the first, may as well involve the origin of the two other nerves, and impair their functions, by modifying the action of their common ganglionic cells. Stilling and Lockhart Clark have shown that the sixth and facial nerves arise from the same nucleus in the oblong medulla. Vulpian has also demonstrated by his interesting researches on the origin of the facial, its intimate connections with the sixth pair in the floor of the fourth ventricle. More recently yet, John Dean, in his valuable work "On the Gray Substance of the Medulla Oblongata and Trapezium,"* alluding to the nucleus from which the facial arises, says: "Several other cell-groups are found, both on the outer and inner side

* Smithsonian Contributions of Knowledge, 1864, p. 66.

of the upper olivary bodies, and very many cells are found scattered throughout the whole anterior and antero-lateral network. Among these groups, the largest and most constant are, one on the inner side of the olivary body in the vicinity of the roots of the sixth nerve, consisting of stellate, multipolar cells of moderate size, and another on the outer side of the olivary body, near the entrance of the facial roots, consisting of quite large multipolar cells, and sometimes, as noticed by Schröder Van der Kolk, forming two distinct groups, the cells of which become more and more numerous, and at the same time are pushed inwards as we reach the upper part of the course of the facial, continuing to increase both in size and number as we approach the fifth nerve, to the motor root of which I suspect this group is related, as well as to the facial." The evidence of these statements suffices to account for the paralytic symptoms, without the necessity of the ingenious hypothesis put forward by Brown-Séquard to explain the nature of reflex paralysis. It may be questioned how such a derangement in the oblong medulla can reach the facial and sixth nerves, sparing those of the limbs. The special influence of the nucleus belonging to each of the above nerves, the narrow limits of the relations just pointed out, as much as the nature of the local excitation restricted to the trigeminal, and the incomplete character of the paralysis, explain why the limbs were not affected, and why, for these very motives—when the disturbance in the circulation of the medulla oblongata

reached its maximum during the epileptic paroxysm, and the trouble became more extensive—the arms were also temporarily paralyzed.

Concerning the rotatory movement of the body during the epileptic attack, it evidently was the effect of the local irritation to the cerebral hemisphere. It is well acknowledged that lesion of the cerebral hemisphere and other regions of the brain originates a rotatory movement of the body, the real cause of which is not yet satisfactorily explained. The deviation of the eye in this case was due to paralysis and not contraction, for I could ascertain its persistence when the head was raised or moved in any direction. It was most interesting to observe, after the epileptic paroxysm and before consciousness was recovered, the rolling or oscillating state of the eyes, reproducing the nygtasmus that accompanies the strabismus and rotatory movement of the body noticed on animals, upon injury of those parts of the brain capable of determining such a peculiar movement. I may add that, in those cases of epilepsy where I have met with nystagmus evident cerebral lesion has also existed.

In conclusion, the case is a curious instance of polyuria due to traumatic lesion of the head. The researches of Bernard, Schiff, Pavy, and others, have brought to light the influence of the vaso-motory nerves in the production of diabetes. From the striking coincidence here of polyuria, converging strabismus, facial palsy, and other trouble with the vaso-motory nerves, I cannot avoid surmising, what is per-

haps a mere speculation, *i. e.*, the possibility of a disturbance in the floor of the fourth ventricle, where is the origin of the nerves involved by the above paralysis, the diabetic centre (*centre diabétique*) of Bernard, and besides the centre of the visceral vaso-motory nerves, as demonstrated by the interesting anatomical investigations of Jacubowitsch. Cases are not rare of injury to the brain, spinal cord and nerves, as also to the sympathetic system, attended with diabetes. It has been remarked, as regards injuries to the head, that this condition happens sooner or later upon the accident, and that it may change most suddenly from diabetes mellitus into polyuria. I have been unable to ascertain if such change did occur in this instance.

I may cursorily allude here to a male epileptic, now at the Hospital and troubled with polyuria. Counter-irritation to the nape of the neck, as practised by Goolden * and Kunkler, † has arrested the diabetic trouble in this instance in a very remarkable manner. The quantity of urine from the beginning diminished from 140 to 60 ounces daily. Struck by the result, and curious to ascertain if it were due to counter-irritation, this was stopped, and the quantity of urine increased the first day to 130 ounces and the next to 136, and decreased again on renewal of the blisters, which have also considerably amended the fits.

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* Medical Times, December, 1854, and Lancet, June 24 and July 15, 1854.

† Union Médicale, 20 Juillet, 1861.

The circumstances in which I have been placed prevent me to estimate without fallacy the degree of mental failure in relation to the nature and duration of the epileptic attacks, or the comparative frequency of epileptic insanity in reference to epilepsy generally. The majority of the Hospital patients, primitively proceeding from the City Lunatic Asylum, have been naturally tainted with more or less insanity. Therefore, I have limited myself, in what refers to the subsequent remarks, to compare only those cases where epilepsy assumed on the onset its characteristic symptoms of unconsciousness and convulsion, to become thereafter a source of well-defined mental derangement, or epileptic insanity. I avail myself of this term, proposed by Falret, as less open to misunderstanding than that of epileptic mania, synonymously employed with furor, which refers to a feature, important it is true, but not essential to the mental derangement of epileptics. Epileptic insanity has come under my close observation in 118 cases, of whom 55 were males and 63 females. I need not repeat that the greater proportion of females proceeds from their having been almost exclusively received at the Hospital, whereas the male department has been more equally divided between epileptics and paralytics.

The nature of the attacks was as follows :—

	MALES.	FEMALES.
Petit mal	7	9
Grand mal	26	25
Petit mal and grand mal	13	18
Nocturnal spasms	9	11

In regard to hereditary predisposition we find—

	MALES.	FEMALES.
Mother epileptic	3	4
Mother insane		2
Mother intemperate		1
Father insane		2
Father paralyzed	1	
Father epileptic and intemperate		1
Father intemperate	4	
Maternal uncle epileptic	1	3
Maternal uncle insane	1	1
Paternal uncle epileptic	2	
Paternal uncle insane	1	
Parents first cousins; intemperance and nervous disease in her antecedents		1

The causes were acknowledged twenty-one times among males and fifteen among females.

	MALES.	FEMALES.
Mental disturbance	1	
Fatigue	1	
Grief		1
Fright		1
Seeing a playmate in a fit		1
Insolation	1	
Syphilis	1	
Injury to the head	3	1
Intemperance	16	7
Childbirth		1
Pregnancy		1

The age of the disease was:—

	MALES.	FEMALES.
Childhood	10	13
Adolescence	14	29
After 25 years	31	21

The foregoing data show hereditary predisposition more frequently derived from the mother's than from

the father's side, and also entailed by intemperance. Mental derangement supervened more frequently in relation to the grand mal, and nearly in the same proportion to the petit mal and nocturnal attacks. The characteristic paroxysms were exhibited at some of the stages in every case; yet, the convulsive or the vertiginous phenomena did not attract attention constantly, insanity obviously constituting by itself the malady in such instances, irrespective of any other sign of epilepsy. The frantic agitation of epileptic delirium is universally known: the brain becomes thus deranged, suddenly, without intermission, after the spinal system has been set into the disordered action originating convulsions. This is, indeed, a peculiarity to be kept in mind. The reflex faculty of the spinal cord cannot react several times in succession without exhausting itself, hence the brief duration of the epileptic spasms, seldom lasting over one or two minutes. Not so with the cerebral activity, almost continuous, and, therefore, capable of being disordered by the epileptic fit in a transient manner, or persisting over a period of several hours or days. The furor, as already advanced, is by no means essential to epileptic insanity; ordinarily, I have met with it upon repeated attacks, and it is so characteristic, that it can hardly give rise to doubt of any kind as to its diagnosis. Insanity, however, displays itself in other forms than furor, dementia, or imbecility. To diagnosticate this mental derangement, when not connected with spasms, is not always easy, and may

involve highly important medico-legal points. Epilepsy contrasts singularly with other diseases in the deep marks it impresses on the organic and moral constitution of the individual. Scarcely do we find confirmed epileptics who, on careful examination, would not afford evidence of this statement. The intellectual impairment originates by degrees, or by imperceptible stages,—nay, it may be tardy in its display, though it affects the instincts and emotions from the beginning. Epilepsy draws to depravity—the fact evinces itself whether we look at the victim of the disease in any of the social ranks. The change is undergone more rapidly by him who breathes an atmosphere of vice than by him who does not. Social surroundings must unquestionably exert their influence. In the first instance the patient becomes irritable and overbearing, a liar, addicted to masturbation, or other wicked habits; whereas, in the second case, the seed sown in abundance yields a more pernicious fruit, and drunkenness, larceny, arson, murder, or crime in its most hideous conceptions, lead the victim by counted steps to the penitentiary or the gallows. Believing that there is a morbid obliteration of moral feeling in epileptics, and that they are mastered by uncontrollable impulses, I would hesitate to fix the limits of their responsibility. Chronicity to my mind is as essential as any of the other characteristics of epilepsy, wherefore I would be far from thinking a person epileptic because he or she were troubled at some previous time with epileptiform convulsions that

have not reappeared. Consequently, I reject exculpating any overt act attributed to epilepsy, unless committed in clear relation with the spasms or while the perpetrator exhibited plain signs of cerebral epilepsy. And, whichever be the medico-legal faith given to this last form of the disease, it is nevertheless positive that epileptics undergo a moral degeneracy through their malady, and that we are never safe with them, as it has been recently asserted with great truth by Delasiauve.

Follet, Baume, Morel, and others pointed out a disproportion between the cerebral hemispheres of epileptics, idiots, and imbeciles. L. Duchesne, as previously remarked, noticed unilateral hypertrophy of the brain among the accidental, but not constant, phenomena of epilepsy. Bourneville has further advanced, upon thirty-three autopsies, that the difference of weight between the cerebral hemispheres is in the majority of cases none or very slight, and not related to the intensity and frequency of the fits. Delasiauve has equally found the disproportion almost imperceptible in five or six cases. As I previously stated, my own experience in this respect bears out most fully the unilateral changes of the brain—hypertrophy and sclerosis—as well as a disproportion in the weight of the cerebral hemispheres, which has neither been constantly in one side, nor always extremely great, though yet considerable enough to be taken into account. To complete my researches I have taken the shape of the skull of the epileptics at the hospital, with the *con*

formateur used by hatters, in order to ascertain whether an unsymmetrical or peculiarly conformed cranium is exhibited by epileptics. The skull has been found irregular in every instance; but I have ascertained that this is a peculiarity common with most human skulls. No fair criterion can be established from the tracings I have collected. The unsymmetrical figure has, however, been more striking in those affected since infancy, or tainted with hereditary predisposition to epilepsy. Tracings, reduced of two-thirds from the original, are here given, fig. 1, of the head of an epileptic since infancy, and, fig. 2, of one with hereditary



Fig. 1.



Fig. 2.—(F. Forehead.)

epilepsy. The cranium measured, in the former: circumference, 21½ inches; antero-posterior diameter, from root of the nose to occipital protuberance, 13½ inches; transversal diameter from one to another auditory meatus, 13 inches. In the latter case: circumference, 21¾ inches; antero-posterior diameter, 13¾

inches; transversal diameter, 14¼ inches. The smaller the dimensions of the skull the greater has been found the irregularity of the head: the fact is most manifest in fig. 3, showing the head of an epileptic imbecile, with circumference 20½ inches, antero-posterior diameter 12½ inches, and transversal diameter 13 inches. Finally, fig. 4 represents the head of a female epilep-



Fig. 3.



Fig. 4.

tic, who reunited in her case almost every hereditary cause of degeneracy. The dimensions were: circumference, 20¾ inches; antero-posterior diameter, 12¼ inches; and transversal diameter, 13¾ inches. The father and mother of this woman were first cousins. A great-aunt, an aunt, and several cousins on the mother's side, a great-uncle, an uncle, and a cousin on the father's side, were epileptic. Her father, her father's brother, a brother, and sister are hard drinkers. Her father was very passionate, and, under liquor, killed her infant brother and sister (twins). Her brother and

sister died of phthisis. She became epileptic upon childbirth, the attacks were always nocturnal, and followed by fits of mania the day after. She is most troublesome, frequently fighting with her companions, and exhibits a most extraordinary coincidence of so many agencies of human degeneracy.

Insanity complicates ordinarily the advanced stages of epilepsy, although it may set in periodically from the very first paroxysm, as in five of the cases under consideration. Hospital records show the mental derangement chiefly connected with oft-repeated but not severe attacks, or with that state of relapsing convulsions similar to the *status epilepticus* of French alienists. I could not assert that the duration of the disease influenced in any constant manner the development of insanity, for this is registered as frequently with recent as with old standing cases, and equally at all ages. Prolonged arrest of fits has told with severity on the maniacal excitement, it being further curious that nocturnal attacks were comparatively the most apt to induce mental impairment. It is throughout the apparent lucidity of epileptic insanity that patients give vent to their sudden unprovoked violence. The mere sight of a person, a trifling remark, calls forth an outbreak of their morbid irritability, and instead of a convulsive paroxysm, an assault or a fit of fury takes place. I could mention several examples establishing this point of extreme medico-legal value, and will bring forward the following :—

CASE XXXV.—*Nocturnal Epilepsy from Injury to the Head—Epileptic Insanity, with homicidal impulse.*

G—— B—— was admitted into the Hospital for Epileptics at the end of October, 1869. His father was intemperate, his mother died with phthisis, and one of his brothers is also epileptic. He was a printer, is now aged twenty-five, has been moderately temperate, and enjoyed good health till July, 1864, when he received a blow on the head from a cog-wheel of a printing-press. The blow fell upon the right parietal bone, involving the suture, and, to a slight extent, the left parietal bone, also leaving a linear cicatrix of the scalp, without extending in any noticeable manner to the bone underneath. The accident rendered him insensible for about ten minutes, but did not prevent his resuming his work. For about four months afterward he suffered from slight but pretty constant headache, aggravated by drink. His first fit occurred while at work, a few months after the injury. The night previous he had indulged more than usual in drinking. Three months later he had a second nocturnal fit; and after it the attacks recurred at intervals of about two months, always with their nocturnal character, and becoming more frequent, one every two weeks, about nine months before admission into the Hospital. He has since then had three fits in November, one in December, one the 1st of January, and two the 20th of March—all of them at night. I must add, that I became cognizant of the last two fits only on the 20th of April, through one of the night-watchmen, who informed the Assistant Physician of it. This patient is at times of quick, irritable temper, and has acted as a helper, taking no medical treatment whatever since last January. For two nights previous to the 30th of March, he was discovered going slyly to tumble a helpless paralytic out of bed for the sake of fun. On the morning of the 30th of March, he was quietly addressed by another patient, whereupon he became furious, violently assaulting and striking brutal blows at his companion and severely injuring his left eye. He was not at all excited immediately after this act of violence, and continued quietly his work of setting the table for the patients' breakfast. He offered no excuse for the assault, other than "that he could not help it, being greatly provoked at his companion's remark." The sudden explosion of madness in this instance supplanted the epileptic paroxysm; and neither before nor directly after the sudden attack of furor did this epileptic give proof of insanity.

Case XXXVI.—*Nocturnal Epilepsy attended with Polyuria and homicidal furor.*

M—— D—— is a sailor, aged thirty-eight. His father was paralyzed. He has nocturnal fits often, many times in succession in one night, almost always preceded by a cloudy or stupid condition, which leaves him in a talkative and incoherent state. He lately exhibited distressing symptoms of polyuria, to which I have already alluded, and which were stopped by blisters to the nape of the neck. One morning this epileptic, without any provocation whatever, seized a knife near by and assaulted one of his companions, who narrowly escaped being stabbed. The mental trouble in this instance is not apparent to any person who would not inquire closely into the patient's symptoms; scarcely does he show any agitation, and he obstinately conceals his feelings and hallucinations of a terrifying nature.

Case XXXVII.—*Epileptic Insanity and Suicide.*

C—— B——, aged thirty-one. His father was intemperate and passionate, and his mother died in convulsions on giving birth to him. He had infantile palsy with atrophy of the right limbs and divergent strabismus of right eye. He became epileptic at the age of twenty-three, the fits being preceded by an aura starting from the bladder and stomach. The spasms more frequently occurred at night, and he could control them by emptying his bladder. He was a masturbator, of uncontrollable temper, though seemingly very humble, and would wander constantly around the grounds of the Institution. The fits decreased in frequency, while he became morose and silent. One morning, appearing more disturbed than usual, he left with another patient a letter in which he disposed of his effects, stating that he departed for a better place, and went and drowned himself in the river. The suicidal impulse substituted in this instance the more common homicidal tendency observed with maniacal epileptics.

Numerous facts, recorded by alienists, evince that epileptic maniacs, though unconscious of their acts, may exhibit integrity of thought, and power to speak during the epileptic paroxysms. Trousseau says in unequivocal terms: "Every physician, who has studied

epileptic vertigo practically, must have seen cases of individuals speaking and answering questions during the attack—speaking, it is true, in a strange, jerked voice, but still answering questions *to the point.* The paroxysm once over, they have no recollection of what has just passed." * Many of our patients at the Hospital, during their paroxysms of epileptic mania, lasting three or four days, quarrel and fight with nurses, assistants, or companions, speaking in the most abusive and intentional manner, so as to make any person ignorant of their respective condition believe they are conscious of their acts. A girl, aged 22, would use the most abusive language after her fits, biting her attendants, and breaking everything. She has often rushed to throw herself out of the window, and once assaulted and seriously injured her younger sister. All these cases confirm, that power to speak should not be safely admitted as evidence of criminality when the loss of moral freedom is suspected to be due to epilepsy.

The less apparent the convulsions, the more perplexing becomes the question of epileptic insanity. Falret has minutely detailed the phenomena proper to this psychical condition. An impulsive want to wander about, a vague sense of anxiety, and groundless terror, a constant automatic motion, picking up everything near at hand, a miserable feeling of intolerable inward sensations, are precursors of the sudden invasion of fury—from which the epileptic immediately comes out with dim recollection of what has just oc-

* Clinical Medicine, vol. i., p. 26.

curred. The indistinctness of recollection may be erroneously looked upon as simulated, but, as Falret remarks, it is perfectly real and characteristic of this mental condition. Another typical feature, which Falret places in a first rank, is, that when laboring under the insanity in question, epileptics say that they are no longer themselves, that they are overwhelmed by evil spirits, and that their will is driven on. A female at the Hospital, with epileptic vertigo, has attempted frequently to commit suicide, and exhausts her rage by knocking her head against the walls of the cell, and crying loudly without ceasing. This woman says that when she feels the fit approaching, "she asks God to deliver her from it, and that she cannot disobey the voice that draws her to what she does." A lady recently sent to me by my friend Professor Ch. A. Budd, has been troubled with petit mal and grand mal. The disease has existed for several years unsuspected, in the form of nocturnal fits, probably commencing at the age of puberty. This lady has slight attacks of vertigo through the day, preceded by a vision of a sudden dash of fire like lightning. She is also disturbed in her sleep by dreadful dreams. Her memory has failed, and she complains of not being herself, and feeling insane without a will. She has been seized with blind impulses to strike her infant child, and being unable to resist it, her mother has been obliged to take the child away from her.

The intellectual derangement may precede the epileptic paroxysm, as it did happen in 13 males and

17 females of the patients here considered. Generally mania supervenes upon the attack, the furor lasting from an instant to several days. No disagreement exists as to this point with those conversant with epilepsy. It is not, however, established, and I maintain it upon repeated observation, that epileptic insanity sets in but never passes off suddenly, and that it continues with intermittent exacerbations for days or weeks in succession, when not as a persistent condition. The language and deportment of epileptics in this state of alienation bear a striking character of irritability and quickness, or of sadness and dulness, which contrast strongly with the automatic execution even of the most indifferent acts. The epileptic at this stage is not master of deliberating or choosing: he starts according to his most pressing feeling, completely powerless to resist it, and thus may be unconsciously drawn to criminal deeds. The insidious, violent, unprovoked perpetration of these latter display their epileptic origin, which under the circumstances becomes of great moment.

The frequency of hallucinations is greater than suspected. I have noted them distinctly in 92 out of 116 cases of epileptic insanity. They were usually unacknowledged by the patient, who described them only when pressed on the subject. Hallucinations of hearing existed in 61 cases; in 83 others the patients saw sparks, visions, or brilliant objects before their eyes. Only thrice have I met with hallucinations of smell in females. Anæsthesia, partially extending over some of the limbs, existed in 5 males and in 11 females.

Furor may herald or follow the convulsive fits, but it may again replace them altogether under the form of periodical mania or cerebral epilepsy, masked epilepsy, *épilepsie larvée*, of Morel. This masked form I have frequently met with in relation to intemperance and injuries to the head, as in Case XXXIV., where the incubation of the disease was probably favored also by hereditary predisposition.

The following are further instances of this curious exhibition of epilepsy:—

CASE XXXVIII.—*Epilepsy—Attacks assuming a Cerebral Form.*

M. D——, aged 36, epileptic from grief since the age of 22, is an old hospital patient. She had three fits respectively, on the 12th and 13th of May, and remained as usual insane, running around the room, and shouting unintelligible sentences. This excitement subsided partially in two days, when she ran into a comatose condition, with pulse at 100, and firm, lasting almost five days. Turpentine injections relieved and roused her, but she did not completely recover her intellectual faculties until after a convulsive fit, on the 20th of May. Since then the spasms have disappeared, or rather been replaced by periodical mania, preceded by a semi-comatose state.

CASE XXXIX.—*Epilepsy—Attacks degenerating in absences, with Religious Monomania.*

A gentleman, aged 35, subject every two weeks to convulsive fits attended with mania for two or three days, has improved so much that he has had lately only one spasmodic fit in nine months. The attacks, however, are replaced by sudden absences, during which he remains unconscious for an instant, though yet able to continue with what he is doing. He has been troubled besides with delusions and religious mononania. He says that the fits of absence afford him a great relief, for he feels distressed and confused in his mind when they do not occur in several days.

CASE XL. *Hereditary Epilepsy—Epileptic Mania—Attacks preceded by Intellectual Brightness.*

A young man, aged 22, with inherited predisposition to epilepsy in his father's and mother's family, is partially demented after his fits. He is also a monorchid. Previous to the attacks, always nocturnal, he displays a remarkable remembrance of events that happened long before, and talks in a bright manner, that disappears immediately after the paroxysms. He has been seized with an obstinate determination to run away from the house, and to give himself to attack violently those near him, but otherwise appears of a gentle and kind disposition.

I wish to state once more, before closing this chapter, that I have not pretended to dwell on epileptic insanity in all its bearings; but simply to bring forward the striking points connected with it, as displayed by the foregoing cases. The subject is of cardinal importance, and entangled with difficulties. Its prominence demands wider limits than those of these last pages, where I have simply anticipated, in a brief manner, what I will soon consider more fully in a work on Epileptic Insanity, which will be the complement of these researches.

APPENDIX.

I may add some brief remarks on the subsequent course of the three cases before referred to, in which I resorted to trephining of the skull for the relief of epilepsy. I received information that for the last two years and a half (August 5, 1870) no recurrence of fits has taken place with the first patient, Case XXXII., and, therefore, his cure appears to be permanent. The other two died not long ago. The one whose history is reported in Case XXXIV., left the Hospital in the beginning of last January, with the head wound completely cicatrized, and in a sound condition of mind, to resume his former occupation of carriage-driver. He died June 25, 1870, and I learnt from Dr. T. H. Trippler, who attended him, that he exhibited again maniacal paroxysms, though without convulsive fits, on different occasions for a short time before his death. On post-mortem examination Dr. Trippler found the bone surrounding the trephined portion of the parietal natural; intense cerebral congestion; brain substance indurated and elevated underneath the perforation of the left parietal bone; and general superficial sclerosis (?) of the gray substance. Old effusion of lymph forming a layer around the caver-

nous sinus at the anterior and posterior lacerated foramina, and around the vessels and nerves at the base of the brain. Longitudinal fissure glued together down to the corpus callosum; fissure of Sylvius in the same condition. Cerebellum giving indications of former inflammation of the membranes. Cerebral ventricles healthy looking, without effusion. No lymphatic exudation on, or induration of, the medulla. Other viscera not examined.

The above cadaveric appearances reveal the existence of peri-encephalitis, probably superinduced by the irregular habits and occupation of the patient, before he had recovered sufficiently from the cerebral derangement caused by the traumatic injury to the head, which was, however, relieved by the removal of the diseased portion of the left parietal bone, when the patient left the Hospital.

The other patient, Case XXXIII., continued all the time at the Hospital. I cannot account, indeed, for the cause of the insidious cerebral trouble, as he has been particularly watched, and seemingly progressing towards recovery. He had his last fit during the second week in May, and was taking thirty grains of bromide of potassium in addition to counter-irritation to the neck, when, in the beginning of last July, he became melancholic before gradually falling into an unconscious condition, with pulse 96 and feeble, cheeks red, pupils dilated and slow to contract, tongue furred, temperature 102, and urine and excrements passed involuntarily in bed. Quinine and stimulants im-

proved his condition; yet a relapse took place soon, with profuse general perspiration, greater debility, very stertorous breathing, and delirium. The bromide of potassium was then discontinued, July 20, 1870, and he steadily sunk thereafter, until he died in a comatose state in the morning of the 27th of July; no fits having occurred since last May, or for nearly three months.

Post-mortem examination was made twenty-four hours after death, by Drs. G. J. Swirchevski and A. E. Macdonald, Assistant Physicians to the Hospital. Body emaciated. Skin of the scalp over the right temporal region infiltrated with exudation, the periosteum in this place separating very readily from the bone. Cranium regularly shaped and of ordinary thickness. No other traces of cranial fracture beyond that sustained by the right parietal bone, extending nearly parallel to its squamous portion, from the mastoid angle upwards to the anterior border. A perforation, fully three inches long by more than half an inch wide, marks the portion of the posterior fragment of the parietal bone removed by the operation. This extensive opening was closed by fibrous exudations adhering to the dura-mater. The cerebral membranes looked normal over the hemispheres, but the meninges were yellowish, opaque, and friable (fatty) on the right side, underneath the lower extremity of the fracture, and attached to the dura-mater forming a cystic cavity at this place. This cyst was filled by a red semi-transparent mass, the size of a large

hazel-nut, continuous with some ramifications of the posterior and middle meningeal arteries, completely occluded throughout a long portion of their traject. The cerebral substance at this site was united to the meninges, and the right supra-marginal lobule and ascending parietal gyri were puckered and evidently atrophied, though firm and otherwise unchanged as to their external appearances. No exudation, or unnatural vascularity on any of these regions, and the cicatrix tissue around the cyst evinced that the cerebral substance had been deeply lacerated at the time of the skull fracture. The main lesion was located at the base of the brain, covered with a thick film of opaline lymph, changing into a dirty greenish color over the pons Varolii and the medulla oblongata. The cerebral tissue under this extensive layer, masking the origin of the cranial nerves, was almost diffluent, and, like the rest of the brain, in a far advanced stage of putrefaction, hastened by the excessive heat at that time. Tissue of the cerebellum and medulla not softened. Capillary congestion throughout the cerebral cortical substance and centrum ovale. The cerebral blood-vessels appeared uninjured, excepting the above-mentioned branches of the right meningeal arteries. No pathological change observed in the thoracic or abdominal viscera, beyond some atheromatous patches in the aorta and adhesions of the left pleura to the thoracic walls.

Three different portions of the brain tissue, one of them digested in chloroform, were carefully analyzed

by Mr. J. Murphy, Druggist to the Hospital, and each test determined the unequivocal existence of bromine in the cerebral tissue, seven days after the patient had stopped taking bromide of potassium. Namias has recently shown that bromide of potassium, after its administration, can be detected in the brain, liver, lungs, and in the blood. The fact here noticed, further indicates that bromine may remain accumulated in the brain as in the kidneys, which, as pointed out by Namias, and as I have often observed, may eliminate through their secretion the bromine for several days after the administration of the bromide has been suspended. This phenomenon merits attention on account of its importance in establishing the actual physiological effects of the bromide of potassium.

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